SYNOPSIS OF MEDICINE

PREFACE TO THE FIRST EDITION

THIS book aims at providing a synopsis of such principles of medicine as are of importance at the present time.

A wider scope has been adopted than merely the classification of the most prominent details of each disease. So far as possible the symptoms have been fully enumerated and briefly explained, and the pathology of the disease and references to the most probable or best-known theories have also been included. At the same time it is hoped that, by means of short summaries and special headings, those data which are of greatest importance have been clearly indicated.

The sections on treatment have been planned to afford a ready reference to a reasonable procedure, and no attempt has been made to give numerous alternative methods or prescriptions.

A full index has been provided.

It is hoped that the book may be of assistance to those who have to revise rapidly their knowledge of medicine in general or of some disease in particular: to the worried student whose final examinations are within sight and to the hurried practitioner from whose ken they have long passed, possibly even to the teacher with a lecture to prepare and to the examiner who, for the purposes of a *viva voce*, desires to renew for a brief period his knowledge of any of the essential details of medicine.

The 'synopsis' cannot replace a text-book to the student, and any attempt to make it do so will inevitably lead to failure.

The general arrangement of the book follows that of Osler's universally known *Principles and Practice of Medicine*, and for their kind permission to do this our special thanks are due to the publishers, Messrs D. Appleton and Company. Exceptions to this occur in various portions of the book, and many alterations and additions have been made. The section on Diseases of the Nervous System has been rearranged in accordance with the advice of a well-known neurologist. Considerable changes have also been made in the sections on Diseases of Metabolism, of the Alimentary System, of the Blood, and of the Circulatory System.

I have frequently referred to and am indebted to numerous

works, especially to the large systems of Allbutt and Rolleston, and of Osler and McCrae, and to the monographs of Judson Bury on *Diseases of the Nervous System*, of Rolleston on *Diseases of the Liver and Gall-bladder*, of Lewis on *Clinical Disorders of the Heart-beat*, and of Mackenzie on *Diseases of the Heart*. Among others I have also referred to Manson's *Lectures on Tropical Diseases*, Daniels' *Tropical Medicine and Hygiene*, Muir and Ritchie's *Manual of Bacteriology*, Campbell Thomson's *Diseases of the Nervous System*, Sequeira's *Diseases of the Skin*, Panton's *Clinical Pathology*, and Warren's *Text-book of Surgery*. Other sources have, I trust, been acknowledged in the text.

The special arrangement of headings and types is on the same system as in Hey Groves' *Synopsis of Surgery*, to which this was planned to be a companion volume.

A great amount of time and trouble has been spent on its preparation. The numerous subheadings and types have involved heavy labour for the publishers, and the great number of facts and theories included has necessitated much revision. As the result of this, and of a long interruption due to the War, the book appears several years after the original date assigned, and its publication has repeatedly been delayed, even since the return of peace: yet it is inevitable that many passages must still occur which need or would benefit from alteration, and any criticisms and suggestions will be welcomed.

Finally, I must thank the publishers—and especially Dr. A. E. Mahood and Mr. F. S. Hunter, who have read the entire proofs—for the great care they have bestowed upon the production, for their kindness in waiting for the manuscript until I was able to return to civilian practice, and for numerous suggestions and comments, which have saved many errors that otherwise would have escaped notice.

Dr. Panton has also read certain portions of the manuscript and helped me with his advice.

H. L. T.

LONDON.

July, 1920.

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SYNOPSIS OF MEDICINE.

Section I.—SPECIFIC INFECTIOUS DISEASES.

A. BACTERIAL DISEASES.

CHAPTER I.

TYPHOID FEVER.*

An acute disease due to infection by *B. typhosus*, characterized clinically in typical instances by: (1) Fever; (2) Rose-coloured eruption; (3) Enlarged spleen; (4) Abdominal tenderness; (5) Diarrhœa or constipation.

The symptoms and degree of severity are very variable. Marked localization may occur, especially in lungs and central nervous system.

ETIOLOGY.

General Prevalence.—Typhoid fever is the most common continued fever in temperate climates, but exists throughout the world, without notable differences. *Death-rate* in England and Wales in 1910, 46 per million persons. Is greater in other countries.

Season.—Most prevalent in autumn: probably due to effect of temperature on existence of organisms outside the body.

Sex.—Males and females equally liable. In hospitals more frequently seen in males.

Age.—Most frequent in youth and early adult life, between ages of 10 and 30. At extremes of life, course tends to be atypical. Infants rarely attacked. Is never congenital. Very rare over 50 years of age.

Immunity.—One attack usually protects.

BACTERIOLOGY.

Morphology.—Short, thick, actively motile bacillus with rounded ends. Involution forms, often of great length, common, especially in old cultures. No spores. Flagella, 8 to 12 in number: need special stains. Stains with all ordinary stains, but is *Gram-negative*. These characteristics are common to the coli-typhoid group.

Growth optimum at 37° C. Cultures killed at 60° C. in thirty minutes. Resistant to drying.

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* Typhoid fever is here described as it occurs in uninoculated persons. The variations in inoculated individuals and in paratyphoid infections are referred to at the end of the section.

The term 'typhoid' is now sometimes confined to infections with *B. typhosus*, 'enteric' including also paratyphoid infections. This is often convenient, but the terms are still in general use as synonyms.

Typhoid—Bacteriology, *continued.*

Cultural Characters.—Grows readily on all usual media. On solid media growth usually appears moist. No gas produced in any carbohydrate media. Special characters: (1) Lactose, dulcite, saccharose: no change. (2) Dextrose, mannite, maltose: acid, but no gas. (3) Litmus milk: acid, but no clot (after ten days often returns to alkaline). (4) No indole formation. (5) Gelatin: no liquefaction. (6) Neutral-red broth: no change, or slightly yellow. (7) MacConkey's medium: yellow colonies.

Selective Media.—Numerous media have been devised for the growth and isolation of *B. typhosus.* These media depend mainly on reactions of various dyes. Their value results from: (1) Differentiation of *coli* and *typhosus* colonies; (2) Inhibition of *coli* group and enhancement of *B. typhosus.* Most commonly used are: *MacConkey's* medium—neutral-red bile-salt peptone lactose agar ('rebipel-agar'). *Conradi-Drigalski*—crystal-violet, nutrose, lactose, and other constituents. *Fawcus'* brilliant-green and picric-acid medium. *Browning's* brilliant-green method—peptone-water (5 c.c.) containing 0·5 c.c. of 1–10,000 solution of brilliant-green; method is based on inhibitory action on the *coli* group. *Ox-bile*: typhoid group grows very readily.

Differentiation of Coli Group.—*B. coli communis*: (1) Produces red colonies on MacConkey's medium; (2) Produces acid and gas in lactose and most carbohydrates; (3) Acidifies and clots milk. Also other differences. *B. proteus* produces yellow colonies on MacConkey, but liquefies gelatin.

'NON-LACTOSE FERMENTERS'.—The pathogenic bacilli, typhoid, paratyphoid, dysentery, do not ferment lactose. *B. coli* and most of the non-pathogenic bacilli ferment lactose, but some only slowly ('late lactose-fermenters'), and *certain common strains* of the *coli* group do not at all, and must be differentiated by (1) agglutination, and (2) other cultural characteristics.

MAC CONKEY'S MEDIUM.—'Non-lactose fermenters' form yellow or colourless colonies; *B. coli* and lactose fermenters form red colonies.

Methods of Isolation.—These are equally applicable to paratyphoid bacilli, but brilliant-green is strongly bactericidal to dysentery bacilli.

1. FROM THE BLOOD.—Cultures into broth or ox-bile. Incubated one to five days. Identification of any growth.
2. FROM STOOLS OR URINE.—Cultures into broth (incubate two hours) or Browning's brilliant-green peptone water (incubate twenty-four hours). Plate on MacConkey's medium.
3. FROM THE SPLEEN.—Best method at autopsy. Remove spleen entire. Cut with sterile knife. Cultures into broth or brilliant-green. Plate on MacConkey's medium.

Numerous variations of the above methods are in extensive use.

Distribution of Bacilli in the Body.—

1. ACUTE STAGES.—(1) *In blood*: present for first 5 days, not subsequently. (2) *Peyer's patches* and intestinal lymphoid

tissue: after first few days until ulceration occurs; may then be present deeper in wall. (3) *Spleen*: most numerous and easily isolated, but also present in kidney and other solid organs. (4) *Gall-bladder*: often in large numbers. (5) *Fæces*: probably invariably present after first few days. (6) *Urine*: present in small proportion in later stages.

2. CHRONIC FORMS (*see also below*, CARRIERS).—(1) *Gall-stones.* (2) *Pus and typhoid abscesses.* (3) *Excreta of 'carriers'*, usually fæces. The gall-bladder is the main reservoir for chronic forms.

The bacillus has been isolated from numerous sites—from lungs in pneumonia, from endocarditis, from rose-spots (rarely), etc.

Survival of Bacillus outside the Body.—

IN WATER.—In sterile water cultures of bacilli live many weeks. In natural waters uncultured bacilli, from excreta, die in less than two weeks. In aerated water, bacillus lives not more than two weeks. Infection has resulted from ice.

IN MILK.—Lives and multiplies without changing the milk's appearance.

IN SOIL. Can live several months. Probably does not multiply.

IN STOOLS AND SEWAGE. Dies in three to five days.

ON CLOTHES AND MATERIALS. May live many months.

B. typhosus fulfils Koch's postulates: (1) Is constantly present in the disease, (2) Can be isolated and cultivated outside the body in successive generations; (3) The isolated organism reproduces the disease.

MODES OF CONVEYANCE OF INFECTION.

The bacilli are discharged in the excreta, which directly or indirectly are the cause of spread.

1. **Contagion.** Local propagation, mainly by fingers, food, and flies. Direct transmission through the air is extremely improbable.

2. **Infection of Water.**—Contamination of water-supply is usual cause of large epidemics. Often due to defective sanitation.

3. **Typhoid Carriers.**—Bacilli may persist for years in the body without symptoms after typhoid fever. Found in *gall-bladder* and gall-stones, fæces, intestines, and bone abscesses after 20 years and upwards. No limit to length of time. Numerous outbreaks have been traced to typhoid carriers, especially to cooks, bakers, and dairy employees.

   Women form three-fourths of carriers (in peace).

   Serum usually gives marked agglutination reaction, but not invariably.

   Stools frequently contain numerous typhoid bacilli.

   Bacilli may be present in stools of persons who have had no symptoms of typhoid fever. Especially true in children.

4. **Infection of Food.**—Outbreaks have been traced to several articles: *Milk*—contamination by infected water, or typhoid carrier; *Ice, vegetables, salads*; *Oysters*—certain outbreaks, e.g., Winchester. *Any article handled by a typhoid carrier may convey the bacillus.* (*See also* FOOD POISONING.)

Typhoid—Conveyance of Infection, *continued.*

5. **Flies.**—Power to carry bacillus is certain. In South African War, constituted an active agent in spread.

6. **Contamination of the Soil.**—*Bad sewers or cesspools* are predisposing causes, and may lead to infection of water-supply. *Dust* may carry bacilli. Bacilli when desiccated die rapidly.

MORBID ANATOMY.

Intestines.—The changes characteristic of typhoid occur in the lymphoid tissue of the intestines, mainly in Peyer's patches, *especially in the last foot of the ileum.* Also in solitary follicles. Condition is a proliferative inflammation followed by necrosis. Four stages: (1) *Hyperplasia*; (2) *Necrosis and sloughing*; (3) *Ulceration*; (4) *Healing and cicatrization.*

1. HYPERPLASIA.—Swelling of Peyer's patches and the solitary follicles.

   Commences with hyperæmia, followed by hyperplasia, viz., increase of lymphoid and epithelioid cells. Follicles and patches project above the surface. Blood-vessels compressed, hence projections are often greyish. Condition at maximum from eighth to tenth day.

   Necrosis is usual result. *Resolution* may occur in mild cases, by degeneration of cells and absorption without ulceration.

   Similar hyperplasia is seen in children with intestinal affections, also occasionally in measles, diphtheria, and scarlet fever. In adults, very rare apart from typhoid fever.

2. NECROSIS AND SLOUGHING.—Necrosis of swollen lymphoid elements, resulting in formation of sloughs.

   Depth of necrosis varies. In solitary follicles superficial. Deepest in patches near ileocæcal valve. Usually involves submucosa; may perforate peritoneum.

   Reticulated appearance of a Peyer's patch frequently caused by independent necrosis of several follicles.

   The necrosis may be the result of the action of toxins produced by the bacilli, or due to blockage of blood-vessels.

3. ULCERATION.—Separation of sloughs.

   Sloughing commences at edge of necrotic area. Extent and depth depend on necrosis. Uncommon to affect entire Peyer's patch.

   TYPHOID ULCER results from separation of slough.

   Characters.—*Long axis* in line of intestine. *Shape*: usually irregular oval. *Edges*: soft, undermined, swollen, not indurated. *Floor*: smooth; usually formed of muscularis. *Peritoneal surface*: changes slight.

   Patches often in various stages in different parts of intestine.

   Ulcers most frequent and numerous in last twelve inches of ileum.

4. HEALING AND CICATRIZATION.—Granulation tissue forms and covers floor. Epithelium then extends inwards from edge of mucosa. Glands may re-form partly.

   Healed ulcer is smooth, slightly depressed, and pigmented.

Finally, practically no sign of scar remains.
Stricture never follows, and intestinal obstruction never results.
Majority of deaths occur before cicatrization commences.

Typhoid bacilli are present in tissues in early stages, but diminish or disappear during necrosis.

LARGE INTESTINE.—Lymphoid elements affected in one-third of cases. Severity diminishes with distance from ileocæcal valve. Occasionally is extensively affected, and then often severe in sigmoid and rectum, with marked changes in ileum.

PERFORATION OF THE BOWEL (*see* SYMPTOMS).

HÆMORRHAGE FROM THE BOWEL.—Results from separation of the slough. Blood present in intestine.

Mesenteric Glands.—Hyperæmia and, later, hyperplasia and swelling occur as in intestinal lymphoid tissue. Necrosis and absorption follow. Foci of necrosis common. Glands in mesentery at lower end of ileum especially involved. Suppuration very rare. Rupture of gland extremely rare: may cause peritonitis or fatal hæmorrhage.

Spleen.—Enlarged invariably in early stages. *Increase moderate.* Weight over 1½ lb. uncommon. Soft consistency. Changes similar to glands: hyperæmia and, later, hyperplasia, returning to normal about fourth week. Rupture very rare. Infarcts not common. Typhoid bacilli scattered throughout, often in clumps.

Bone-marrow.—Changes very similar to those in lymphoid elements.

Liver.—Hyperæmic. Swollen in early stages only. Some parenchymatous and fatty degeneration of liver cells. Foci of leucocytes and sometimes of lymphoid cells not uncommon. Typhoid bacilli frequently present. Liver abscess extremely rare.

Gall-bladder.—Cholecystitis may occur, but rare. (*See also* CARRIERS.)

Kidneys.—Cloudy swelling usual. Acute nephritis occasional. Rarely miliary abscesses. *B. typhosus* and *B. coli* may be present.

CYSTITIS occasionally occurs: due to *B. typhosus* or, more commonly, *B. coli.*

Respiratory System.—

LUNGS.—*Bronchitis practically invariable in early stages.* Following also occur: (1) *Lobar pneumonia*, early or late in the disease: in 5 per cent of fatal cases. (2) *Hypostatic congestion* and splenization: late stages in feeble patients. (3) *Hæmorrhagic infarction.* (4) *Fibrinous pleurisy*: empyema rare. (5) *Gangrene and abscess of lung*: occasional termination of pneumonia.

FAUCES.—Ulceration of larynx due to presence of typhoid bacilli occurs rarely. Occasionally: œdema of glottis; diphtheroid conditions of pharynx and larynx.

Circulatory System.—

HEART.—Endocarditis and pericarditis rare. Ulcerative endocarditis usually due to pyogenic organisms, but typhoid bacilli have been isolated. *Myocarditis* not infrequent: muscle soft, pale, and flabby. Fatty and granular degeneration common. Zenker's hyaline degeneration rare.

Typhoid—Morbid Anatomy, *continued.*

BLOOD-VESSELS.—Thrombosis of veins, especially left femoral, not uncommon complication. Changes in the arteries are slight.

Nervous System.—Organic changes rare. Meningitis extremely rare.

Voluntary Muscles.—Zenker's hyaline degeneration may occur. Condition not confined to typhoid fever, but very rare in other febrile states. Affected muscles may rupture. Abdominal muscles, adductors of thigh, and pectorals most common.

SYMPTOMS.

A general description of the symptoms is given here. In the next section are considered the modes of onset, and the special features and symptoms, complications, and sequelæ, according to the various systems.

An ordinary attack of typhoid fever is generally described as consisting of: period of incubation; period of onset; the febrile period, divided into, and referred to as, the first, second, third week, etc. (usually three weeks); and convalescence. The changes in the symptoms, and the complications and sequelæ, often agree with these periods very closely; but they must not be considered as hard-and-fast divisions.

Period of Incubation.—Commonly 10 to 15 days. Ordinary limits 5 to 23 days. Extreme limits 3 days (culture swallowed) to 4 weeks and upwards.

Symptoms of lassitude commence, and period merges into next stage.

Period of Onset.—*Onset is insidious.* Very rarely abrupt.

INITIAL SYMPTOMS.—(1) *Headache*: most common symptom, persistent and severe. (2) Weakness and languor. (3) Abdominal pain. (4) Diarrhœa or constipation. (5) Anorexia. (6) Epistaxis. (7) Chilly sensations (definite rigors uncommon). All these are common.

Symptoms become more severe. Patient takes to bed. Date of onset and division into weeks is usually reckoned from day of taking to bed, or estimated from temperature chart.

First Week.—(1) *Appearance*: Cheeks flushed; eyes bright tongue furred. Slight deafness common. (2) *Headache* rarely absent. May be slight mental confusion. (3) *Bronchitis* almost invariable; crepitations at both bases; cough usually slight. (4) *Abdomen* tender and slightly distended. (5) *Bowels*: Diarrhœa or constipation. (6) *Temperature* rises steadily by 'steps.' On evening of fourth day reaches 103°. (7) *Pulse*: (*a*) *Rate slow compared with temperature — in adults rarely exceeds* 105; (*b*) Low tension—more commonly *dicrotic* than in any other fever. (8) *Between seventh* and *tenth day*, three important events occur: (*a*) *Spleen* becomes palpable; (*b*) *Rash* appears; (*c*) *Agglutination reaction* becomes positive.

Second Week.—Mental torpor. No headache. Expression dull. Pale face, with occasional flush, dilated pupils, and dry lips form characteristic appearance. Deafness often marked. *Temperature*

remains constantly high. *Pulse* may remain slow: usually becomes more rapid; no longer dicrotic. *Tongue* dry. *Abdominal symptoms* increase. Constipation obstinate. If diarrhœa, stools resemble 'pea-soup'. Delirium in severe cases, especially at night. Death may occur with pronounced nervous symptoms. Hæmorrhage and perforation may occur towards end of week.

Third Week.—*Period of dangerous complications.* In ordinary cases, general symptoms remain as in second week. *Loss of flesh* and *weakness* now marked. *Temperature* becomes irregular, with morning remissions, and commences to decline. *Pulse* 110 to 130.

UNFAVOURABLE SYMPTOMS.—(1) *Mental symptoms* pronounced: 'typhoid state' or delirium. (2) *Temperature* remains high or rises. (3) Cardiac weakness: pulse very rapid or irregular. (4) Pulmonary complications: pneumonia, hypostatic congestion. (5) Extreme weakness.

SPECIAL DANGERS, due to separation of sloughs.—(1) Hæmorrhage. (2) Perforation.

In mild cases, symptoms subside.

Fourth Week.—

IN ORDINARY CASES.—Convalescence commences: *Appetite* returns; often ravenous. *Temperature* gradually becomes normal. *Tongue* cleans. Mental and abdominal symptoms subside. General condition is extremely weak.

IN SEVERE CASES.—General aggravation of symptoms. 'Typhoid state' may occur: Face cyanosed; clammy perspiration; dry fissured tongue; sordes of lips; delirium, muttering or frequently restless, or 'coma vigil'; incontinence of urine and fæces; lungs congested; rapid, feeble pulse, often irregular.

SPECIAL DANGERS.—Failure of heart. Secondary complications.

Fifth and Sixth Weeks.—In ordinary cases, general progress. In protracted cases, convalescence commences. *Relapses, recrudescences, complications,* and *sequelæ* may occur.

SPECIAL FEATURES AND SYMPTOMS.

Modes of Onset.—Onset usually insidious. *Localization of symptoms* to one system not uncommon at onset: extremely deceptive, and diagnosis difficult: onset may be acute in these types.

The variations from the normal onset may be grouped thus:—

1. WITH PULMONARY SYMPTOMS.—Clinical types: (*a*) *Lobar pneumonia*: pneumo-typhoid: commonest form of localization. (*b*) *Acute pleurisy*: pleuro-typhoid. (*c*) *Bronchitis*: exaggeration of common initial bronchitis.
2. WITH NERVOUS SYMPTOMS.—Clinical types: (*a*) *Headache* of exceptional severity. (*b*) Facial neuralgia. (*c*) Delirium, especially in ambulatory forms. (*d*) *Mania* and mental symptoms. (*e*) Symptoms of cerebrospinal meningitis, rare, mostly in children, occasionally simulating basal meningitis.
3. WITH GASTRO-INTESTINAL SYMPTOMS.—Clinical types: (*a*) *Acute gastritis.* Incessant vomiting. (*b*) *Appendicitis.* May be closely simulated. (*c*) *Diarrhœa.*

Typhoid—Special Features and Symptoms, *continued*

4 WITH ACUTE NEPHRITIS

5 AMBULATORY OR LATENT FORMS —Patient may 'fight the disease' and remain at work until symptoms and signs of the second week' are present Subsequent course often very severe Delirium common Mortality high Rarely, *perforation* or *hæmorrhage* from the bowels is first symptom

Facial Aspect.—In first week eyes bright and cheeks flushed Later, with mental torpor, the eyes become dull

Fever.—

1 TYPICAL COURSE —

STAGE OF ONSET AND FIRST WEEK —Temperature rises by 'steps' the evening rise being about 2° and the morning fall about 1° Approaches 104° on evening of fourth day Fastigium (maximum) at end of week usually about 104°

SECOND WEEK — Temperature steady Daily variations slight

THIRD WEEK —Temperature becomes remittent and falls by 'steps' The morning temperature shows increasing falls for two or three days while the evening rises to its previous height Then the evening temperature also falls progressively

Evening temperature reaches normal in fourth week morning temperature a few days previously

2 VARIATIONS DURING ACUTE STAGES —

a TEMPERATURE OFTEN HIGH WHEN FIRST OBSERVED 'steps in rise having occurred previously

b RAPID RISE to 103° 104 may occur with rare initial rigor, or with lobar pneumonia or localization of symptoms

c SUDDEN FALL important Occurs with (i) *Intestinal hæmorrhage* rapid anæmia collapse (ii) *Perforation* rises again as peritonitis develops, pulse rapid Rapid but not sudden, fall occasionally with severe nervous symptoms

d IN MILD CASES, temperature may fall rapidly in second week, or be abbreviated or modified to all degrees

e IN SEVERE CASES, febrile period may persist for many weeks

f RISE DURING COURSE may occur with (i) Increasing severity, (ii) Lobar pneumonia or other complications Hyperpyrexia above 106°, of serious prognosis

ASPIRIN usually causes sudden fall followed by rapid rise

3. POST-TYPHOID VARIATIONS —

a RELAPSES AND RECRUDESCENCES (*see* RELAPSES p 20)

b PERSISTENT FEVER DURING CONVALESCENCE — Evening rise may persist several weeks in weak patients In absence of symptoms, and of bacilli from excreta, may finally be disregarded

c PERSISTENT HYPOTHERMIA DURING CONVALESCENCE —In weak patients Is of no significance Subnormal morning temperatures common

Rigors.—Not common. In general, a rigor suggests a complication, and repeated rigors an error of diagnosis. Occurrence .—

(1. AT ONSET.—Rare. Repeated rigors, very rare.

2. AT INTERVALS throughout the febrile stage. Rare. May be sweating ('sudoral form'), and simulate malaria. Occasionally severe rigors in later stages, ascribed to slight sepsis.
3. AT ONSET OF COMPLICATIONS.—Pneumonia, pleurisy, and venous thrombosis; occasionally with perforation or hæmorrhage.
4. WITH ANTIPYRETIC DRUGS.
5. OCCASIONALLY AFTER BATHS AND SPONGING.

Rash.—

TIME OF APPEARANCE. Seventh to tenth day.

FREQUENCY.—About 70 per cent of cases. Less frequent in children.

SITE.—Abdomen and chest commonest, then back and thighs. Face, hands, and feet very rare.

CHARACTERS. Rose-red, slightly raised, flattened papules. Disappear entirely on pressure, and reappear rapidly on release.

SIZE. 2 to 4 mm.

NUMBER.—Usually scanty and widely scattered. Frequently less than a dozen. Appear in successive crops, persist about three days, then fade, leaving slight brownish stain. Number of spots bears no relation to severity of attack.

VARIATIONS IN THE RASH.—

Occasionally very profuse.

Rarely, appears first in a relapse, or after subsidence of fever.

Purpuric spots may occur. Very rarely a true hæmorrhagic typhoid fever.

Spots occasionally vesicular.

OTHER ERUPTIONS.

MACULÆ CERULEÆ or PELIOMATA.—Occur with rash. Slate-coloured spots, about twice the size of rash. Always scanty; usually on thighs, abdomen, or chest. Are caused by lice: ascribed to pigment in the salivary glands of the louse: occur, though rarely, in other febrile conditions.

SUDAMINA and MILIARIA. Not infrequent with sweats.

ERYTHEMA.—Occasionally in first week. May occur independently of drugs.

HERPES.—Very rare.

Skin—Various Lesions.—

ODOUR.—Of 'abdominal' character in severe cases.

SWEATS.—Skin usually dry. Sweats may follow cold baths, and occur with venous thrombosis, hæmorrhage, or perforation. Occasionally repeated sweats and rigors throughout course.

BEDSORES.—In severe cases tend to form rapidly.

ŒDEMA.—May result from: (1) Venous thrombosis; (2) Anæmia and weakness—bilateral; (3) Nephritis—very rare.

NOMA and GANGRENE OF SKIN.—Very rare.

BOILS.—Not uncommon, but usually in convalescence. Frequent after cold baths. Obstinate, but rarely dangerous. Due to streptococcus or staphylococcus, not to *B. typhosus*.

HAIR.—Often falls out during convalescence of severe cases.

Typhoid—Skin Lesions, *continued.*

usually avoided if hair cut short in early stages. Grows again as before. Permanent baldness very rare.

LINEÆ ATROPHICÆ.—May resemble results of pregnancy.

Blood Changes.—Changes in leucocytes of diagnostic importance.

LEUCOCYTES. (1) *Leucopenia* throughout course. Frequently under 4000. (2) *Lymphocytes relatively increased. Polynuclear leucocytosis* occurs with peritonitis or septic complications.

ERYTHROCYTES AND HÆMOGLOBIN.—Progressive secondary anæmia. Rarely severe until third week.

Changes persist into convalescence, and gradually disappear.

Circulatory System.—The most important phenomena are :—

FOR DIAGNOSIS.—(1) Pulse-rate relatively slow and often dicrotic in first week ; (2) Blood-pressure low and tends to fall. Also leucopenia with relative lymphocytosis.

FOR PROGNOSIS.—Rapidity and irregularity of pulse.

COMPLICATIONS. Venous thrombosis. Cardiac weakness.

SEQUELÆ.—Disordered action of the heart.

THE PULSE.—

FIRST WEEK.—(1) *Rate* : In adults, rarely exceeds 105, even with high fever. Usually 85 to 95. This relatively slow pulse is very constant and of importance in diagnosis. In children more rapid. In severe cases with high temperature, may be rapid throughout : prognosis serious. (2) *Character* : Dicrotic pulse common.

SUBSEQUENT WEEKS.—Frequently more rapid, 110 to 130, and smaller, but may remain slow throughout. *Not dicrotic.*

CONVALESCENCE.—Bradycardia common, about 50. May be extreme, but is of no importance. Rarely tachycardia.

THE HEART.—

HEART SOUNDS usually normal. Cardiac dullness not increased.

MYOCARDITIS in severe cases : feeble first sound, and soft systolic murmur audible at apex and pulmonary area. In grave conditions, irregularity, etc., occur, as in cardiac exhaustion : of serious prognosis.

ENDOCARDITIS and PERICARDITIS rare. Latter mainly in children or with pneumonia.

DURING CONVALESCENCE.—Any form of DISTURBANCE OF THE CARDIAC RHYTHM (q.v.) may occur, especially after severe attacks, or with predisposing causes. Rare.

BLOOD-PRESSURE.—Characteristically low. Systolic pressure in earliest stage 110 to 125 mm. Hg. Falls in second week to 90 to 100 mm., remaining low until convalescence. With hæmorrhage may be rapid fall. Rises with perforation.

THROMBOSIS OF VEINS.—One of the common complications.

FREQUENCY.—In 2 to 3 per cent.

TIME.—During third week or later. Not uncommon when temperature normal : may follow a slight exertion.

SITE.—Lower extremity, with few exceptions. Left femoral vein forms 50 per cent of cases. Probably predisposed to by pressure of right iliac artery.

SYMPTOMS.—(1) Rise of temperature. Rigor common: may be sweating. (2) Sudden pain at and near site. May be partial degree of collapse, suggesting perforation. (3) Thrombus usually palpable (examine gently). (4) Swelling of extremity follows. (5) Leucocytosis usually present.

PROGNOSIS.—Good. Death from pulmonary embolism very rare, with proper treatment. Tendency to slight œdema prolonged, and varices frequent. Gangrene very rare.

PULMONARY EMBOLISM from small emboli probably not very rare: dyspnœa, pain, and slight signs in lungs.

CAUSE uncertain. Coagulability of blood may be increased.

THROMBOSIS OF ARTERIES. Very rare. Extremity becomes blue and pulseless. Recovery usual. Gangrene rare.

Digestive System.—

APPETITE.—Lost early. Returns with convalescence, and becomes ravenous.

THIRST.—Constant. Must be gratified.

TONGUE.—Furred. At onset, thin moist fur, which gradually thickens. In ordinary cases dry in second week. In mild cases, moist throughout. Cleans in fourth week or in convalescence. Saliva diminished. *In severe cases*: (1) Tongue dry, with brown fur; (2) *Sordes* on teeth and lips. Mouth must be kept clean.

PAROTITIS.—Rare, but mortality high. Frequency 1 per cent. Chiefly in third week of very severe cases. Very rare when mouth properly treated. Usually unilateral. Generally suppurates; may be extensive sloughing. Origin: probably extension of inflammation along Steno's duct.

CANCRUM ORIS.—Very rare. Only in children. Is painless, and commences on mucous surface. Recovery exceptional.

PHARYNX.—May be congested. Membranous pharyngitis: rare, only in cases otherwise serious. Typhoid ulceration not known.

ŒSOPHAGUS.—True typhoid ulceration may occur: very rare. Dysphagia. Stricture may follow.

GASTRIC SYMPTOMS. Slight. Nausea and vomiting rare. Occasionally severe and obstinate at onset. After first week, vomiting extremely rare, and suggests complications—e.g., peritonitis, nephritis. Hæmatemesis very rare: of septic origin.

Abdominal Symptoms.—

GROUPS.—(1) Pain and tenderness; (2) Distention and meteorism; (3) Diarrhœa; (4) Constipation; (5) Spleen; (6) Hæmorrhage; (7) Perforation of ulcer; (8) Liver.

PHENOMENA OF MOST IMPORTANCE.—

FOR DIAGNOSIS.—Abdominal tenderness; palpable spleen; diarrhœa and 'pea-soup' stools.

COMPLICATIONS.—Hæmorrhage; perforation of ulcer.

FOR PROGNOSIS.—Severe diarrhœa; marked distention; and above complications.

SEQUELÆ.—Gall-stones; 'typhoid carriers'.

Typhoid—Abdominal Symptoms, *continued.*

1. ABDOMINAL PAIN AND TENDERNESS.—

   TENDERNESS AT ONSET usually present. Pain rarely severe. Diffuse, umbilical, or in right iliac fossa.

   PAIN DURING COURSE less common. Constantly with peritonitis, rarely with hæmorrhage, occasionally with diarrhœa or constipation, or with pleurisy, thrombosis, or distended bladder. Cause frequently indefinite.

2. ABDOMINAL DISTENTION.—*Meteorism or Tympanites*: Due to loss of tone of muscular coats of intestine or stomach. Moderate degree common, and of little importance: on palpation feels doughy. *If severe*, prognosis bad, distention impedes heart and lungs and favours perforation: occurs also in peritonitis. Gurgling in cæcum very common: of little importance.

3. DIARRHŒA. *Note*: Diarrhœa with 'pea-soup' stools is a characteristic symptom, but occurs in less than 50 per cent of cases, and with modern avoidance of purgatives is considerably less common. Profuse diarrhœa is most common in severe cases.

   TIME OF ONSET AND DURATION.—Present from beginning, and persists throughout in about one-third of cases. May develop in second week. May alternate with constipation.

   CAUSE.—Catarrh of gut, especially of large intestine. No relation between diarrhœa and extent of ulceration.

   NUMBER OF STOOLS.—Varies: three to ten daily.

   CHARACTER OF STOOLS.—Thin, large quantity. At first of normal colour; after a few days comparable with 'pea-soup'. Reaction alkaline. Odour offensive, especially in children. On standing, separation into two layers, fluid above, semi-solid below. *Mucus scanty.* Shreds from sloughs are very rarely recognizable. Defæcation painless. Typhoid bacilli rarely present before end of first week. Milk curds, a sign of defective digestion, must be watched for. (Stools of closely similar nature occasionally occur in children apart from typhoid fever.)

4. CONSTIPATION.—As frequent as diarrhœa, or more so. May occur with advanced ulceration. (Mortality in general is lower in cases with constipation than with diarrhœa.)

5. SPLEEN.—Becomes palpable at beginning of second week in 70 per cent of cases. Especially in children. In elderly patients not so constant. Gradually subsides during third week. Palpable area small: often only tip. Recognition of enlargement by percussion alone is uncertain.

6. HÆMORRHAGE.—Serious and important complication.

   FREQUENCY.—In 6 to 7 per cent of cases. Incidence increases with age, rarer in children.

   TIME OF OCCURRENCE.—*Between end of second and beginning of fourth week*, the time of separation of sloughs. In 'ambulatory' type, may be first definite symptom. (*Note*: Slight hæmorrhage from congestion may occur in first week, unimportant except for diagnosis.)

SYMPTOMS.—Slight hæmorrhage may occur without symptoms, except melæna: important, as severe hæmorrhage often follows. In severe hæmorrhage, symptoms are:—

a. Sudden onset: without warning.

b. Sensation of faintness, followed by pallor and symptoms of collapse. Restlessness, sighing respiration, cold sweat, vomiting, and, with large hæmorrhage, rapid anæmia. *Pain variable*, absent or severe. No distinctive physical signs in abdomen.

c. Rapid fall of temperature: frequently subnormal.

d. Pulse small, rapid and running.

e. Blood-pressure falls, often 80 to 90 mm. Hg.

f. Stools, bright blood or tarry. Passage often delayed from hours to one or two days after hæmorrhage. Death may occur before passage.

g. Quiet delirium or mental 'wandering' is common.

Hæmorrhages not uncommonly repeated: may be numerous.

Leucocytosis occasionally present.

PROGNOSIS.—Always serious. Single hæmorrhage rarely fatal. Mortality, about 20 per cent with repeated or profuse bleeding. Peritonitis from perforation may follow (in about 20 per cent). Cause of death in 5 to 10 per cent of fatal cases.

PERFORATION OF TYPHOID ULCER.—

FREQUENCY. Occurs in 3 to 4 per cent of cases. Causes 25 per cent (at least) of deaths from typhoid. Commoner in men than women.

AGE.—Rare over 40 years, and in young children.

TIME OF OCCURRENCE. Usually in third week. Very rare earlier. Not infrequent in fourth or even fifth week if pyrexia persists. Very rare when temperature normal.

SITE.—Usually in ileum; commonly within 12 inches of ileocæcal valve. Occasionally in sigmoid and appendix. Rarely in other sites. *May be several perforations.*

CAUSE OF PERFORATION. *Separation of sloughs*: slough often adherent to edge of perforation. Perforation may be pin-point, or, less often, extensive from separation of large slough. Errors of diet, purgatives, sudden movements of body, etc., are oft-quoted exciting causes, but with present-day careful treatment are rarely present. Rupture of gut from intestinal distention is extremely rare. Necrosis of peritoneum or drag of adherent slough is cause almost invariably.

PREVIOUS COURSE OF ATTACK.—*Usually severe*: particularly with diarrhœa and with tympanites: associated with hæmorrhage not uncommonly. May occur in mild attacks.

SYMPTOMS.—Three stages often recognizable: (*a*) Shock immediately on perforation; (*b*) Latent period or 'period of repose'; (*c*) Symptoms of general peritonitis.

a. Symptoms on Occurrence of Perforation.—

i. *Sudden severe abdominal pain.* In lower abdomen, usually in or near right iliac fossa. Generally in paroxysms.

Typhoid—Abdominal Symptoms, *continued.*

ii. *Sudden change* in constitutional and local conditions, with signs of shock. *Temperature* often falls temporarily: *pulse and respiration* more rapid: cold sweats: occasionally vomiting: may be *rigor. Abdominal tenderness* marked: may be local rigidity and muscular spasm: movement diminished. Blood-pressure rises. Bowels may be moved slightly.

b. Latent Period.—Above symptoms often subside in one to two hours, and for a period of a few hours, while peritonitis develops, practically no symptoms may be present. *May be extremely deceptive.* Period of repose not always present, or incomplete, and the preceding and following stages merge.

c. Symptoms of General Peritonitis (q.v.) *develop.*—If temperature has fallen initially, it usually rises again rapidly. Leucocytosis usually present: important in diagnosis, but may be absent.

DIAGNOSIS.—Usually not difficult. From: (*a*) *Hæmorrhage*: abdominal symptoms slighter, and blanching. May be very difficult, and may co-exist. (*b*) *Appendicitis*: difficult, but differential diagnosis unimportant. (*c*) Phlebitis of iliac veins: very rare. (*d*) *Peritonitis* from other causes: very rare—e.g., rupture of mesenteric gland, typhoid septicæmia, or inflammation spreading through gut. (*e*) Intestinal colic: attacks may occur during convalescence, usually associated with constipation.

With extreme toxæmia, perforation may occur without symptoms, and be found at autopsy.

TREATMENT.—Immediate operation. Mortality very high, but diminishing with early diagnosis and improved surgery.

8. LIVER.—Lesions rare.

ACUTE CHOLECYSTITIS.—Symptoms: Pain, tenderness, and rigidity over gall-bladder. May be a tumour. Jaundice not constant. Result: recovery, suppuration, or rupture and peritonitis. Typhoid bacilli may be isolated in pure culture. Cholecystitis from typhoid bacilli may occur many years after attack.

GALL-STONES.—Not during attack, but subsequently occur more frequently in persons who have had typhoid, owing to persistence of typhoid bacilli in the gall-bladder.

JAUNDICE.—Definite jaundice is very rare.

ABSCESS OF LIVER.—Extremely rare. Due to secondary pyogenic infections. Never from typhoid bacilli.

Respiratory System.—

GROUPS.—(1) Epistaxis; (2) Bronchitis; (3) Lobar pneumonia; (4) Pleurisy; (5) Hypostatic congestion. Less common: (6) Bronchopneumonia; (7) Pulmonary embolism; (8) Laryngitis.

PHENOMENA OF MOST IMPORTANCE.—

FOR EARLY DIAGNOSIS.—Bronchitis; epistaxis.

COMPLICATIONS.—Lobar pneumonia in second or third week; hypostatic congestion.

1. EPISTAXIS.—Frequent early symptom: occurs in about 20 per cent: commoner in typhoid than in any other fever. Rarely serious.
2. BRONCHITIS.—Important in diagnosis. *Presence at onset almost invariable. Physical signs: Crepitations at bases.* Symptoms slight, cough rarely troublesome and complaint rare. Lessens in second week. Not specially marked in fatal cases.
3. LOBAR PNEUMONIA.—May occur at two stages:—
   a. AT ONSET.—*Rare.* Illness may commence as typical lobar pneumonia: more frequently onset somewhat insidious.
      Progress: Defervescence usually does not occur, but occasionally there is a crisis, with subsequent rise. Clinical aspect alters. Pulmonary symptoms subside gradually, and intestinal symptoms become prominent. Condition may become typical of typhoid, or spots and agglutination reaction may decide diagnosis. In absence of spots diagnosis often difficult until late in course. Empyema apparently never follows.
   b. AS COMPLICATION IN SECOND OR THIRD WEEK.—Considerably commoner than at onset. Occurs in 2 to 3 per cent of cases and in 5 per cent of deaths, case-mortality being about 30 per cent. Typical symptoms and rusty sputum usually absent: condition recognized by rapid respiration, cyanosis, physical signs, and often increased pyrexia. Occurrence of 'typhoid state' during ordinary lobar pneumonia must not be confused with these conditions.

   ETIOLOGY is doubtful. Typhoid bacillus frequently isolated from lung at autopsy, and from lung puncture during life. Pneumococcus probably always present.
4. PLEURISY.—Not common: in 1 to 2 per cent. May occur:—
   a. AT ONSET.—Illness commences apparently as acute pleurisy, and, later, clinical condition alters, as with lobar pneumonia. Usually fibrinous: effusion is rare, and pus is extremely rare.
   b. AS COMPLICATION IN LATER STAGES, most commonly during convalescence.—Symptoms of pleurisy less acute, but *empyema usually follows*, and *B. typhosus* may be present in pus.
5. HYPOSTATIC CONGESTION.—Not uncommon in later periods. Prolonged recumbent position renders it frequent in feeble patients. Usually in severe attacks. Symptoms very slight or none, condition being discovered by examination. Physical signs: Impaired resonance at bases, feeble breath-sounds and vocal resonance; râles may be numerous. *Mortality* very high.
6. BRONCHOPNEUMONIA.—Usually only as a terminal event: present in a considerable percentage of autopsies.
7. PULMONARY EMBOLISM.—Rarely, in later stages with venous thrombosis.
8. LARYNGITIS.—True typhoid ulceration occurs, though rarely.

Typhoid—Special Features and Symptoms, *continued.*

Nervous System.—The *mental state* is practically always affected, often profoundly, in all but very mild cases, and frequently for a period subsequent to attack. *In febrile stage* in ordinary forms, there is mental dullness with stupor or mild delirium. Sleep is almost continuous, insomnia being a severer condition. Can be roused without resentment. Mental condition also affected by the headache of onset and by the usual deafness.

The various changes can, in general, be referred to three stages: (1) At onset; (2) Febrile and toxic period; (3) Convalescence.

MEMORY.—*At onset*, memory usually deficient, and patient's record of onset unreliable. Subsequent to attack, all memory of illness often lost, or a hazy recollection of a few incidents. Memory is impaired during convalescence.

DELIRIUM.—Rarely absent in severe cases.

1. At Onset.—Not common. In rare cases, during prodromal period and stage of onset (especially in 'ambulatory' form), dementia or delirium may be earliest symptom; subject may wander far, do strange acts, or even be maniacal.
2. In Febrile Period, during second and third weeks or subsequently. Various types:—
   - *a.* Quiet delirium and stupor. Easily roused temporarily.
   - *b.* Restless and obstinate delirium without violence May attempt continuously to get out of bed.
   - *c.* Low muttering delirium, in severe attacks.
   - *d.* Delirium tremens in drunkards. Apart from this, violent delirium is not common.
   - *e.* *Coma vigil*: Patient lies with open eyes, muttering and oblivious to surroundings. Incontinence of urine and fæces. Tremors of lips, tongue, and limbs. Twitching of fingers (carphologia). Picks at bed-clothes (subsultus tendinum). Is a sign of extreme toxæmia, and mortality very high.

Suicidal tendencies may be present even in mild delirium.

TYPHOID PSYCHOSES.—

1. Delirium and mental changes of onset and of febrile period, as above. (Delusions arising in febrile period occasionally persist into convalescence.)
2. Asthenic Psychoses of Convalescence.—More common after typhoid than other fevers. Some weakening of memory, and even of intelligence, may persist for many months: *full* mental powers frequently not regained under twelve months, but complete recovery with time.
   - '*Post-typhoid Insanity.*'—Dementia and various forms of insanity, such as monomania, may occur. Recovery is almost invariable.
   - '*Post-typhoid Neurasthenia.*'—May continue for months or years and completely prevent mental application. Most severe in neurotic persons, especially if convalescence shortened and return to work hurried; in rare instances, may then be permanent.
   - *Hysteria.*—May occur: rarely serious.

MENINGEAL SYMPTOMS.—

1. MENINGISM.—Symptoms suggesting local affection of meninges may be extremely marked. More common in children. Usually at onset, very rare during course. *Due to congestion*; no gross anatomical lesions present.

Symptoms. Severe headache, photophobia, head retraction, twitching of muscles, and, rarely, convulsions. Facial herpes not uncommon. Meningeal symptoms gradually subside, and those of typhoid develop. Degree of symptoms bears no relation to severity of attack of typhoid fever. At onset, always diagnosed as meningitis.

2. MENINGITIS.—Very rare. Occurs late in disease. Occasionally tuberculous or diplococcal.

CONVULSIONS. Very rare. Causes various. May occur at onset, in children, or may result from meningism, or meningitis.

PERIPHERAL NEURITIS, ETC.—Peripheral neuritis occurs late in disease or during convalescence. Severe pain and swelling in affected area; most frequently extensors of lower extremity.

'TENDER TOES'. Cause doubtful; may be due to local neuritis, or possibly neurasthenia. Tips of toes extremely sensitive to weight of bed-clothes; no swelling.

PAINFUL CRAMPS.—Not uncommon, especially in calves. Probably a myositis, rarely, perhaps, thrombosis of veins.

RARE NERVOUS SYMPTOMS.—

MULTIPLE NEURITIS.—Very rare. During convalescence. Rarely fatal.

APHASIA.—Occurs rarely in children. Prognosis good.

HEMIPLEGIA. Probably due to thrombosis. Aphasia is generally present.

EYE.—Affections very rare. Loss of accommodation occasionally during convalescence. Conjunctivitis, optic neuritis, or retinal hæmorrhages may occur.

EAR.—Temporary deafness almost a constant symptom in early stages. Otitis media in about 3 per cent of cases. Serious results rare.

Renal System.—

CHANGES IN THE URINE.—Common febrile characteristics are present: excretion of chlorides diminished. *Polyuria* frequent during convalescence.

ABNORMAL CONSTITUENTS.—Albumin and casts (*see below*). Acetonuria without glycosuria may occur late, probably from starvation. Glycosuria occasionally during convalescence. (Ehrlich's diazo-reaction is of little value.)

RETENTION OF URINE.—A frequent early symptom. May cause abdominal pain. Suppression of urine is rare.

ALBUMINURIA.—Occurs in following conditions:—

1. FEBRILE ALBUMINURIA.—Occurs in majority of cases. Amount of albumin small. A few hyaline casts may be

Typhoid—**Renal System,** *continued.*

present. Most common in second week. May persist through convalescence. Kidney not permanently affected.

2. NEPHRITIS.—*At onset*: Rare. Uræmia may follow. Diagnosis of typhoid fever very difficult. *During course*: Rare. Most common in second week. In these varieties œdema does not occur. Chronic nephritis does not follow. *During convalescence*: Very rare. Œdema usually present. Chronic nephritis very exceptional.

3. PYURIA.—From cystitis or pyelitis.

BACILLURIA, CYSTITIS, AND PYELITIS.—

BACILLURIA from typhoid bacilli occurs frequently: about 20 per cent. Rarely before third week. Pus or albumin usually present. Urotropine is partial preventive. Persistence after normal convalescence is rare. Cystitis usually caused by *B. coli*; occasionally by typhoid bacillus.

Generative System.—

ORCHITIS.—Rare. During convalescence in young adults. Atrophy unusual.

MASTITIS, OVARITIS, occur rarely.

Osseous System.—

PERIOSTITIS may occur, usually in ribs or long bones. Painful node forms. Occurs in about 1 per cent. May subside or abscess form.

BONE LESIONS.—*Abscess of bones* may occur during convalescence, or more often subsequently, even many years later. Tibia, ribs, and femur most common sites. Onset with severe pain, redness, and swelling. Formation of abscess slow; recovery tedious; recurrence frequent. Pus usually contains typhoid bacillus, either in pure culture or with pyogenic organisms. Thorough surgical treatment is necessary.

ARTHRITIS.—Monarticular or polyarticular. Hip most common. 'Typhoid dislocation of hip' may occur spontaneously.

'TYPHOID SPINE.'—Characterized by severe pain in lumbar and sacral regions. Almost confined to males of 15 to 30 years. Onset during convalescence, frequently after mild attack Generally preceded by aching in back. *Severe pain*, often in agonizing paroxysms. *Spine rigid.* May be no physical signs; usually local tenderness. *Nervous and hysterical symptoms* often present, associated with the pain and insomnia. *X rays*: spondylitis and bony changes present in some cases, but not invariably. Definite spinal deformity may occur, rarely; usually kyphosis. Suppuration never present. Origin may be a periostitis.

TREATMENT.—Complete rest. Immobilization of spine by jacket. Morphia for pain. Subsequent treatment as for fractured spine.

DURATION.—One to twelve months. *Recovery invariable.*

Muscles.—Zenker's degeneration may occur (*see* MORBID ANATOMY).

Post-typhoid Pyæmia and Septicæmia.—Some degree of pyæmia is not uncommon.

FURUNCULOSIS. Often extensive and obstinate. More common after cold baths.

SUBCUTANEOUS ABSCESSES may result from: *General pyæmia* following furunculosis or bedsores — staphylococcic; *Typhoid abscesses* bacillus present in pure culture.

Recurrent chills, late in disease, may be due to slight septic infections.

ASSOCIATION OF OTHER DISEASES AND MIXED INFECTIONS.

Most cases where typhoid fever appears to follow or co-exist with other diseases are errors in diagnosis.

Malaria.—May occur with typhoid, but most cases are either typhoid or malaria, and not both. There is no specific typho-malarial fever.

Influenza.—May co-exist during epidemics. Diagnosis of abdominal influenza from typhoid may be extremely difficult in sporadic cases.

Tuberculosis. The following conditions may be recognized:—

1. Typhoid fever may simulate tuberculosis. Especially at onset with pleuritic or pulmonary symptoms.
2. Tuberculosis may simulate typhoid fever. Especially tuberculous meningitis and acute miliary tuberculosis. More rarely tuberculous peritonitis and tuberculosis of deep lymphatic glands.
3. Tuberculosis, acute or chronic, and typhoid fever may co-exist Tuberculous meningitis occasionally is terminal event in typhoid.
4. Pulmonary tuberculosis may follow typhoid fever. Dublin in New York finds that the death-rate from tuberculosis in the two years following typhoid fever is nearly three times the normal: subsequently rate unaffected.

VARIETIES OF TYPHOID FEVER.

The variation and complexity of the symptoms and course of typhoid fever have resulted in the description of many forms. These depend mainly on the exaggeration, modification, and localization of prominent symptoms. An unusual symptom may be present throughout an epidemic. The more definite varieties are:—

1. **Mild Form.**—Symptoms and course often typical, but greatly reduced in severity. In other cases a selection of typical symptoms. Agglutination reaction usually positive. Diagnosis may only be possible during an epidemic. Patient may not feel ill enough to go to bed. Rarely, the characteristic complications and sequelæ or relapses may occur and first reveal correct diagnosis.
2. **Abortive Form.**—May be a few days' pyrexia and malaise. Frequency with which agglutination reaction is positive is uncertain. Mild and abortive forms may become 'typhoid carriers', excreting virulent bacilli.
3. **Grave Forms.**—Severe nervous symptoms and high fever most frequent. Prostration may be extreme from commencement (adynamic form).

Typhoid—Varieties, *continued.*

Cases with intense localization of symptoms at onset are usually severe—e.g., pneumonic forms.

A general hæmorrhagic form occurs very rarely, with occasional recovery.

4. **Ambulatory or Latent Forms.**—(*See* MODES OF ONSET.)

5. **An 'Afebrile' Form** is described. Extremely rare.

Typhoid Fever in Children.—Presents certain differences from that of adults. Such variations, described below, are most marked in infancy, and diminish up to 10 years of age. After this, disease approximates to adult type.

IN INFANTS UNDER TWO YEARS.—Rare. Diagnosis usually suggested by possibility of infection (as in epidemics) rather than by symptoms. Confirmed by agglutination reaction or isolation of bacillus. Mortality high in cases diagnosed (50 per cent).

IN CHILDHOOD.—Most frequent variations from adult type:—

MORBID ANATOMY.—Intestinal lesions not so marked. Ulceration may be absent. In undoubted typhoid, changes may not exceed those of simple diarrhœa.

MORTALITY.—Lower than adults: about 5 to 10 per cent.

ONSET.—Often sudden. Vomiting is common initial symptom. Condition may resemble other gastro-intestinal disturbances of childhood.

TEMPERATURE.—Initial rise frequently more rapid, curve less typical, duration shorter. Usually higher than in adult cases of same severity.

PULSE.—More rapid, but comparatively slow for febrile disease in children. Dicrotism rare.

RASH.—Less frequent, and is scanty.

SPLEEN.—Nearly always palpable.

GENERAL PROGRESS.—Symptoms milder. Condition usually stuporose. Marked delirium and nervous symptoms, such as 'typhoid state,' rare. Meningitis may be closely simulated.

COMPLICATIONS AND SEQUELÆ.—Rare and mild. Hæmorrhage and perforation rare. So also otitis media. Chorea not uncommon. Temporary aphasia, without organic cause, is peculiar sequel: recovery in few weeks.

Typhoid Fever in the Aged.—Incidence rare. Fever not high and course usually atypical. Pneumonia and heart failure common. Mortality high.

Typhoid Fever in Pregnancy.—Pregnancy gives no immunity. Abortion in 70 per cent.

RELAPSES.

Occur in about 10 per cent of cases. Frequency varies in different epidemics.

1. **Ordinary or True Relapse.**—Occurs after temperature has become normal. Average interval five days: rarely exceeds two weeks. Diagnosed by presence of two of the triad: (*a*) Step-like temperature; (*b*) Rash; (*c*) Enlarged spleen. Relapse usually shorter and milder than original attack, but in rare instances is more severe. May be several relapses, becoming progressively milder. Duration seven to twenty-one days: occasionally longer.

2. **Intercurrent Relapse.**—Occurs before temperature has become normal. Often very severe. Complications not uncommon.

3. **Spurious Relapse, Recrudescence.**—Transient rises of temperature of a few hours' to one or two days' duration are not uncommon during convalescence. Occasionally connected with constipation, too rapid progress with diet, or excitement, but often no obvious cause: possibly a mild septic infection. May be slight malaise or no symptoms.

Recrudescences of temperature due to obvious boils, venous thrombosis, etc., are not relapses.

No satisfactory explanation for relapses is known, the blood at the time being strongly bactericidal to typhoid bacilli. Theories include: (1) Original infection with several strains of bacilli, against one or several of which immunity is not established, and such strain multiplies and causes relapse (Durham); (2) Re-infection by bacilli lingering in the gall-bladder—improbable.

DIAGNOSIS.

Methods of Diagnosis.—Typhoid fever is the most common of all long-continued fevers. There are three groups of data for diagnosis, depending on: (1) Symptoms and signs; (2) Bacteriological examination; and (3) Serological examination.

1. SYMPTOMS AND SIGNS. The manifestations are extremely variable. No one symptom or sign is characteristic, except perhaps the rash. Most suggestive in early stages are: (*a*) Insidious onset; (*b*) Temperature curve; (*c*) Relatively slow pulse; (*d*) Headache; (*e*) Bronchitis. The typical triad of typhoid is: *rash, enlarged spleen*, and *the temperature curve*.

   BLOOD.—Leucopenia with relative lymphocytosis.

2. BACTERIOLOGICAL EXAMINATION. The isolation of *B. typhosus* is conclusive, but often difficult. (*See* BACTERIOLOGY.)

   a. FROM THE BLOOD. Initial stage of typhoid is a septicæmia, and bacilli are present in the blood: after a few days bacilli become localized in internal organs. Bacilli can be isolated from blood cultures in first few days, rarely after the fifth. Earliest absolute proof of disease.

   b. FROM THE STOOLS.—Not present in early days. Almost invariably present later, but isolation not always easy.

   c. FROM THE URINE.—Present in nearly one-third of cases, but only in later stages. Rarely in large numbers.

3. SEROLOGICAL EXAMINATION—AGGLUTINATION REACTION (Widal).—(*a*) Positive reaction is not obtained before the seventh or eighth day; may be delayed further. (*b*) Reaction is positive in 95 per cent, at least, of cases with clinical

Typhoid—Diagnosis, *continued.*

symptoms; it is unknown how often it is positive in 'abortive' forms. (*c*) Positive reaction is occasionally delayed until a relapse or convalescence. (*d*) Positive reaction is extremely rare in conditions subsequently proved not to be typhoid.

DIFFICULTIES MAY ARISE FROM:—

a. Doubtful reactions.—Test should be repeated. May be too early in disease, or due to production of a certain amount of agglutinins occurring in other diseases with high fever, e.g., pneumonia, tuberculous meningitis.

b. Previous antityphoid inoculation.

c. Paratyphoid infections. (*See* pp. 31–33.)

A 'positive reaction' represents an increase of agglutinins not only above the titre of normal serum, but also above the titre which may occur in other diseases. Hence a *quantitative* test is necessary. Numerous satisfactory techniques are in use, the only essential being a knowledge of the standards of agglutination in health, sickness, and typhoid fever for the particular method employed; these vary greatly in different methods. The various techniques are based on two principal methods: (1) Microscopic; (2) Macroscopic or sedimentation test. *For the microscopic method, complete agglutination in dilution of serum* 1-50 *in one hour is a positive result.* The standards for the macroscopic test vary greatly in different techniques.

Differential Diagnosis.—*Difficulties in diagnosis* arise from: (1) Localization of symptoms in special organs at onset; (2) The general symptoms and course. A correct diagnosis is often impossible for some days.

Specific tests are not mentioned in this section.

1. LOCALIZATION OF SYMPTOMS.—Diagnosis has to be made especially from the following conditions:—

a. PNEUMONIA.—*Pneumonia at onset* may completely mask other symptoms, but *an initial pneumonia is a rare mode of onset*, and the common error is diagnosing pneumonia as typhoid.

Bronchitis, a constant symptom, and pleurisy occasionally cause error.

b. MENINGEAL SYMPTOMS.—Lumbar puncture may clinch the diagnosis, by cytology or bacteriology.

c. APPENDICITIS.—Typhoid may commence with constipation and pain in right iliac fossa.

2. GENERAL SYMPTOMS AND COURSE.—

a. TUBERCULOSIS.—The usual error is diagnosing tuberculosis as typhoid: the reverse is less frequent.

Acute General Miliary Tuberculosis.—*Temperature* usually more irregular. *Pulse* more rapid. *Polynuclear leucocytosis* often present. The abdominal symptoms may be closely similar, with constipation and palpable spleen. The pulmonary form is more distinct, with definite dyspnœa and cyanosis. (*See also* MILIARY TUBERCULOSIS.

Glandular Tuberculosis.—Especially of abdominal and deep glands. May simulate typhoid for a period.

Tuberculous Peritonitis.—This may simulate typhoid fever when occurring with acute onset.

Tuberculous Meningitis (q.v.).—Vomiting is frequent early, the abdomen is retracted, and the temperature is irregular. Inequality of pupils and squint are common. *Lumbar puncture* decides the diagnosis.

b. SEPTICÆMIC CONDITIONS.—Note in general: (i) Onset more abrupt; (ii) Temperature less regular; (iii) Pulse rapid from onset; (iv) Sweats and rigors frequent; (v) Leucocytosis common; (vi) Etiological factor may be present, e.g., septic foci; (vii) Progress often rapid.

General Septicæmia or *Pyæmia.*

Otitis Media.

Osteomyelitis.—Local pain and tenderness.

Puerperal Septicæmia. Especially as abortion often occurs in typhoid.

Infective Endocarditis.—May be extremely difficult, but onset and progress usually less rapid. In acute forms, purpura and hæmorrhages common. Blood cultures may be positive. Leucocytosis often absent.

c. GASTRO-INTESTINAL CONDITIONS.—

Gastro-enteritis and *Colitis* of all grades, from transien disturbances to acute infections with dysentery, and food-poisoning bacilli. The difficulty is mainly in the milder forms, severe types being very acute.

Appendicitis.

Various Affections of the Abdominal Glands (rarely)—e.g., tuberculosis, Hodgkin's disease.

d. INFLUENZA.—Onset more acute, and respiratory and upper air-passages more affected. Spleen may be palpable. 'Abdominal influenza' is very rare.

e. MALARIA.—Especially in malignant tertian type.

f. ACUTE EXANTHEMATA.—Rarely difficult, except with mild forms of typhus in certain epidemics (*see* TYPHUS).

g. MALTA FEVER.—No spots. Temperature less regular, pulse more rapid. Shorter course with relapses. Geographical distribution (*see* MALTA FEVER).

h. SPIROCHÆTOSIS ICTEROHÆMORRHAGICA.—Acute onset: early severe jaundice. Jaundice is very rare in typhoid.

PROGNOSIS.

Death-rate.—*In hospitals*, should not exceed 15 per cent. Rate is lowest (5 to 10 per cent) between 5 and 10 years. Generally low towards end of epidemics. Severity of epidemics varies.

Mortality higher in hot weather, in fat people, in women than in men, and very high in ambulatory type and in alcoholics. Increased by any pre-existing disease such as diabetes.

Even in mild cases death may result from hæmorrhage or perforation, or symptoms become severe in third week, or rarely during a relapse.

Typhoid—Prognosis, *continued.*

Sudden death is rare, about 3 per cent of fatal cases. May occur in later febrile stages from cardiac failure, usually in males. In convalescence, generally due to pulmonary embolism.

Special Features in Prognosis.– Serious symptoms are mainly dependent on degree of toxæmia and on complications.

NERVOUS SYMPTOMS. – Any definite delirium is serious. In 'coma vigil', mortality very high. Low muttering delirium with tremor, restless delirium, or delirium tremens also serious. Early onset of nervous symptoms unfavourable.

PULSE.—Pulse-rate constantly over 120 is serious, and prognosis is worse as rate increases. Weakness of first sound is early sign of cardiac failure: a soft systolic murmur has little importance. Irregularity a bad sign. The pulse-rate is a measure of toxæmia. In children, of less importance.

TEMPERATURE. — Hyperpyrexia (over 106°) is serious. High temperature below this without other severe symptoms is of slight importance if not prolonged.

ABDOMINAL SYMPTOMS.—(*a*) *Meteorism*, when marked, is a sign of toxæmia. (*b*) *Diarrhœa*: mortality higher than with constipation.

PULMONARY SYMPTOMS.—(*a*) Hypostatic congestion, and (*b*) late lobar pneumonia, have high mortality.

COMPLICATIONS.—(*a*) Hæmorrhage; (*b*) Perforation: mortality very high. The rarer complications are not often serious.

Of little value in prognosis are: profuse rash, initial bronchitis, dicrotic pulse.

PROPHYLAXIS.

Typhoid fever can be completely stamped out by: (*a*) Recognition of all cases, including typhoid carriers; (*b*) Destruction of all bacilli leaving a patient. Prophylaxis deals with: (1) Control of epidemics, (2) Prevention of direct infection from a patient.

1. **Control of Epidemics.**– Epidemics are spread by, and attention must be directed to:—

WATER-SUPPLY. Defective sanitation causes large epidemics, for a contaminated water-supply only remains infective for a limited period unless the contamination is repeated. During an epidemic, all drinking water and milk must be boiled. Light wine is safe, and mineral water. Also siphon soda-water after fourteen days' standing, but not earlier.

TYPHOID CARRIERS. Especially cooks and dairy employees.

FLIES.

FOOD.—Including milk, oysters, and vegetables.

DIRECT INFECTION.

2. **Prevention of Direct Infection from Patient.**– Stools and urine commonly contain typhoid bacilli. Disinfection is directed toward these and any articles which may be contaminated by them. All excreta must be carefully sterilized before disposal.

STOOLS, URINE, AND SPUTUM.—Empty into covered pail containing antiseptics—e.g., crude cresol (cheapest) or carbolic acid—and leave at least two hours. The carbolic acid should not be more dilute than 1–80 after addition of excreta. The urinals and bed-pans must be washed with antiseptics, and if possible allowed to stand in them until required: the bed-pans may be scalded. Great care is necessary to prevent spilling of urine.

FEEDING VESSELS must be kept apart.

LINEN.—Soak in 1–20 carbolic for two hours, and boil.

NURSES and others must wash their hands carefully after any contact with a patient, and especially before taking food. Scrubbing with a nail-brush is sufficient: perfunctory dipping the fingers in an antiseptic is reprehensible. No one in attendance on a typhoid patient should take any part in preparation of food for other patients.

ISOLATION.—It is justifiable to nurse patients in a private house or a general ward if the rules for sterilizing excreta and washing the hands are carried out with due care.

No patient should be discharged or regarded as non-infective until bacteriological examinations of the stools and urine are negative.

Antityphoid Inoculation.—The method was introduced by Sir A. E. Wright, and its value has been fully established.

TWO INJECTIONS are given at intervals of eight to ten days, the first containing 500 million bacilli, the second 1000 million.

LOCAL REACTION AND CONSTITUTIONAL SYMPTOMS commence in four to six hours and last one to three days. The degree varies greatly in different individuals, some showing marked local reaction, and others constitutional symptoms with little local change. The local reaction is swelling, pain, and redness; when severe, the appearance suggests sepsis, but it subsides in a few days, fomentations easing the pain. The reaction is usually considerably less after the second injection. Risks are negligible.

RESULTS OF ANTITYPHOID INOCULATION. The case-incidence is reduced to one-fifteenth, the course is modified, and the case-mortality does not exceed 2 to 3 per cent.

PROTECTION is high for one year. After this it varies in different individuals, but is often considerable for two years.

PARATYPHOID INOCULATION has a similar value. The usual dose of mixed vaccine is 500 million of *B. typhosus* and 250 million of each paratyphoid: the second injection is double this. The reaction is no greater than for typhoid inoculation alone.

TREATMENT.

This is considered under: (1) General management; (2) Diet; (3) Hydrotherapy; (4) Medicinal treatment; (5) Treatment of special symptoms; (6) Management of convalescence. No treatment will abort an attack, but skilful nursing, hydrotherapy, and avoidance of purgatives and unnecessary drugs will lower the mortality.

1. **General Management.**—The room should contain no unnecessary furniture, be freely ventilated, and maintained at equable temperature, when possible about 60° F. There must be absolute

Typhoid—General Management, *continued.*

confinement to bed until convalescence is established, about three weeks after temperature is normal. The use of the bed-pan is essential. The bed-clothes must be light : one, or at most two, blankets are sufficient, and one pillow. A hair mattress is best. Smoothness of bed-clothes is essential, the slightest crease tending to bedsores in toxic patients. A rubber cloth should be placed under the bed-sheet ; in severe cases, a water-bed. The mouth must be cleansed after each feed. The patient should be turned from one side to the other every few hours when stuporose, to prevent hypostatic pneumonia. Must be sponged all over daily. Catheterize if retention.

2. **Diet.**—The general febrile catarrh of the alimentary tract impairs assimilation of food. Also, *undigested* solid matter may cause hæmorrhage or perforation of ulcers, probably mainly by increase of peristalsis. The risk is less than was formerly supposed, and modern diet is becoming more liberal ; but all articles must be easily digestible. General principles :—

DURING FEBRILE PERIOD.—

a. *Milk* must form the basis of all diets.

b. It is unnecessary to adhere to a strict milk diet as an absolute routine. Additional articles permissible : eggs, custard, junket, mashed potatoes, arrowroot (two teaspoonfuls to a feed of milk). *Sugar* is of special advantage owing to high value in calories, and can be administered as (i) lactose added to milk, (ii) chocolate, (iii) lemonade. Meat extracts are best avoided.

c. These additional articles can be given throughout in mild cases. In cases of ordinary severity, sparingly until temperature commences to fall (except sugar). The diet should then contain, if possible, 2500 to 3000 calories

d. Solid food is unnecessary, but small amounts of thin bread-and-butter and biscuits are permissible in later stages.

e. Diarrhœa needs stricter dieting than constipation.

f. Watch stools carefully : if milk-clots appear, reduce or dilute milk ; if persisting, give whey or peptonized milk for a few days. *For meteorism* : similar diet and omit sugar.

g. *Fluid to be given plentifully.* Should be several pints daily, but not in large quantities at one time. As soda-water, barley-water, or well given as lemonade containing sugar.

ADMINISTRATION OF MILK FOOD.—Three pints of milk daily. Give 5 ounces, diluted with half volume of water, every two hours, day and night. To each feed, add full teaspoonful of lactose. Flavouring of coffee or tea may be added.

If asleep, patient must be aroused carefully (or food may enter larynx) : subsequently will sleep again immediately. *The mouth must be cleansed after each feed* with glycerin and borax, hydrogen peroxide, or weak carbolic acid.

IN CONVALESCENCE.—After temperature has been normal for 3 days, give bread and-butter (if not previously) ; for 5 days, pounded boiled fish ; for 10 to 14 days, minced

chicken. A large diet should then be given, but of simple nutritious articles, and return to a full ordinary diet be gradual.

Alcohol.- Unnecessary as routine. With cardiac weakness or severe nervous symptoms, give whisky up to 10 ounces daily.

3. **Hydrotherapy.**- Is of great value; mortality greatly reduced.

INDICATIONS.- High temperature, nervous symptoms.

RULES FOR PRACTICE. (*a*) Sponging above 102·5°. (*b*) Bathing above 104°, every four hours. A feed should always be given subsequently.

CONTRA-INDICATIONS FOR BATH. Great weakness, irregular pulse, severe abdominal pain, hæmorrhage, peritonitis, and venous thrombosis.

a. SPONGING.—This may be with.—

TEPID WATER. Soothes and slightly tires patient. Sleep follows. Temperature but little reduced. Easily performed.

COLD WATER. Unpleasant to patient. Temperature reduced, but may rise occasionally, owing to closure of peripheral circulation. A substitute for cold bath when latter is impossible in practice.

Sponging should occupy fifteen minutes.

b. BATHING.—Bath may be:-

i. AT TEMPERATURE OF 85°.—Patient shivers. *Limbs and chest must be rubbed during bath.* Duration: 10 to 15 minutes. Temperature in rectum falls 2°, and another 2° after return to bed.

ii. AT TEMPERATURE OF PATIENT, and then reduced by addition of ice. Rectal temperature falls 2°.

Pulse must be watched in bath. If it weakens and becomes irregular, return patient to bed and give stimulants.

Hydrotherapy improves pulse, lessens delirium, promotes sleep, stimulates the kidneys, and by these means reduces mortality. Lowering of temperature is not important result. Relapses and boils are possibly more frequent.

4. **Medicinal Treatment.** The administration of drugs in typhoid fever is dictated more often by ignorance and inexperience than by skill and observation. There is no specific remedy, and drugs should never be employed unnecessarily. Pills should never be given.

PURGATIVES are contra-indicated (*see* SPECIAL SYMPTOMS).

ANTIPYRETICS. Quinine (gr. v.) probably does no harm. Other antipyretics are contra-indicated. Mere reduction of temperature is valueless, and collapse may occur.

INTESTINAL ANTISEPTICS are justifiable, but not necessary, if stools be offensive. Their value is unproved, and the excretion of typhoid bacilli is not diminished. Salol (gr. v, t.d.s.) or β-naphthol and others can be given safely.

HEXAMINE (urotropine), is well given in third week, for action as urinary antiseptic (gr. x. t.d.s.). Valueless unless urine is acid.

5. **Treatment of Special Symptoms.**—

HEADACHE.—If severe, cold compresses to head.

Typhoid—Treatment of Special Symptoms, *continued.*

INSOMNIA.—Sponging or tubbing most efficacious.

DELIRIUM AND EXTREME RESTLESSNESS.—*Hydrotherapy.* Hypodermic of morphia (especially to procure sleep). Patient must be watched carefully.

TOXÆMIA.—*Hydrotherapy.* Water freely by mouth. Whisky, 4 to 10 oz.

HYPERPYREXIA.—Hydrotherapy. Avoid antipyretics.

ABDOMINAL PAIN.—Fomentations or turpentine stupes.

TYMPANITES.—*Diet:* albumen-water or whey; no sugar. Turpentine enema or stupes. If severe, *pituitary extract,* 1 c.c., intramuscularly or hypodermically; may be repeated four-hourly. Passage of rectal tube has only temporary effect.

DIARRHŒA.—Examine stools for milk curds: if present, reduce or dilute milk, or give whey or albumen-water for a few days.

FOR SEVERE DIARRHŒA, exceeding four motions a day: (i) Daily starch and opium (tinct. opii ʒss to ʒj) enema (often valuable); or (ii) aromatic chalk by the mouth (e.g., mist. cretæ (B.P.) or pulvis cretæ aromaticæ). Opium by the mouth is best avoided.

CONSTIPATION.—Never harmful. Enema daily or every second day. Never give purgatives.

INTESTINAL HÆMORRHAGE.—(i) *Rest—must be absolute.* (ii) Ice to suck and ice-bag (properly supported) to abdomen. (iii) Opium. Inject morphia, gr. ¼. (*Note*—The objection to opium is that it may increase meteorism and also mask symptoms of perforation, which occurs in 20 per cent of cases of hæmorrhage. The advantages are that it quiets the patient mentally, and diminishes peristalsis). Liable to bedsores and hypostatic congestion.

IN PRESENCE OF COLLAPSE.—(i) Saline injections. (ii) Stimulants: hypodermic injections of camphor (gr. ij in sterile olive oil ♏x).

PERFORATION.—Immediate operation.

CARDIAC WEAKNESS.—*Hydrotherapy,* alcohol, stimulants.

VENOUS THROMBOSIS.—Absolute rest in bed. Limb elevated on inclined plane and wrapped in cotton-wool. Potassium citrate by the mouth is of no value as a preventive.

BACILLURIA.—Urotropine (gr. x, t.d.s.) in hot water after food.

BONE LESIONS AND ABSCESSES.—Operation must be thorough. Typhoid vaccine should be given.

6 **The Management of Convalescence.**—The progress of convalescence must be slow. The temperature should be taken for at least two weeks after becoming normal. Patient may sit up for a few minutes after ten to fourteen days of normal temperature: further progress will be dictated by his strength.

FOOD.—(*See* DIET.)

RELAPSES.—Treatment as in original attack.

COMPLICATIONS OF CONVALESCENCE.—Constipation: treat by enemata. Diarrhœa: restrict diet; confine to bed; chalk or bismuth by mouth.

PARATYPHOID FEVER.

Paratyphoid fever results from infection with one of the three paratyphoid bacilli. Clinically and pathologically it closely resembles typhoid fever, but tends to be milder. Toxic cases, complications, and fatal results are all unusual. The course is still further modified after inoculation with a mixed typhoid-paratyphoid vaccine (T.A.B.). Under MORBID ANATOMY and SYMPTOMS the common differences from typhoid fever are noted. No useful clinical distinction can be drawn between the three types of paratyphoid fever.

Note.—The term 'typhoid' is now being confined, with convenience, by many authorities, to infections by *B. typhosus*, the term 'enteric' including infections by *B. typhosus* and the paratyphoid bacilli.

History.—

ACHARD AND BENSAUDE, 1896, isolated a 'bacille paratyphique' from two patients after attacks resembling typhoid fever. Now known to be the *B. paratyphosus B.*

GWYN, 1898, was the first to isolate the bacillus from the blood in a case like typhoid, and named it *B. paratyphosus.*

SCHOTTMÜLLER, 1900, isolated two varieties from the blood in investigating a series of typhoid patients.

BRION AND KAYSER, 1901, named these *B. paratyphosus A* and *B.*

BAINBRIDGE, 1912, by use of the absorption-method, differentiated *B. paratyphosus B* from food-poisoning bacilli.

HIRSCHFELD, 1916, isolated *B. paratyphosus C.*

Distribution of Paratyphoid Fever.—Before the European War, para B was present in Europe and America, and probably formed 3 to 5 per cent of all cases of enteric; para A was very rare. In India, para A was comparatively common, and probably formed one-third of all cases of ill-defined continued fever (Firth), while para B was very rare.

During the European War, typhoid was rare, presumably as result of inoculation. Paratyphoid was very prevalent in the Dardanelles: until October, 1915, mainly para B, but subsequently replaced by A. In France, the number of cases of enteric was never large; para B was always commoner than A; typhoid formed less than 10 per cent of all cases of enteric.

Bacteriology.—

MORPHOLOGY and METHODS OF ISOLATION.—As for *B. typhosus.*

CULTURAL CHARACTERISTICS.

1. No change in: *lactose*, saccharose, inulin.
2. Produce acid and gas in: *dextrose*, *mannite*, *dulcite*, maltose.
3. No formation of indole.
4. Action on milk: *B. paratyphosus A*—permanent acidity; *B. paratyphosus B* and *C*—slight initial acidity, permanent alkalinity commencing on third day.

Note.—*B. paratyphosus B* can be distinguished from *B. aertrycke* only by 'absorption' tests. *B. paratyphosus C* is not agglutinated by *B. paratyphosus B* antiserum. These three are identical culturally and morphologically.

Paratyphoid, *continued.*

Morbid Anatomy.—The colon is more frequently affected than in uninoculated typhoid fever. Catarrh of the intestine without actual ulceration may be present.

Symptoms.—The clinical course in rare cases may be identical with ordinary or even severe forms of *B. typhosus* infection. Usually it is considerably milder.

DIFFERENCES FROM 'TYPHOID FEVER.'—The following refer to paratyphoid fever of moderate severity, occurring in persons not inoculated with paratyphoid vaccine: many of the patients on whom these are based had received typhoid vaccine.

1. ONSET.—Often more rapid.
2. RASH.—Occasionally very profuse, with large spots (or small areas) of irregular outline, of deeper colour than typhoid, or sometimes a bluish tinge, not entirely fading on pressure, and leaving a slight stain: may almost resemble measles.
3. TEMPERATURE.—Rise more rapid: often 104° to 105° in a few days. Course more irregular, and sustained fastigium unusual. Fall more rapid. Duration about two weeks.
4. PULSE.—Frequently very slow throughout.
5. SPLEEN.—Enlargement may be marked. May be tender.
6. SWEATING and SHIVERING more common.
7. TOXÆMIA rare. Patients with temperature of 104° and a profuse rash often exhibit no toxic symptoms or psychical disturbance, and feel well after first few days.

DIARRHŒAL AND DYSENTERIC ONSET.—Slight diarrhœa not uncommon at onset. Instances occur with acute onset and diarrhœa of dysenteric or food-poisoning type. These only occur in sporadic cases, paratyphoid never producing an outbreak of such type, alleged occurrence being due to confusion of paratyphoid and food-poisoning bacilli (*see below*).

Paratyphoid C.—May produce typical enteric. Has also been isolated from diarrhœal, pulmonary, and various septic conditions without enteric symptoms (Dudgeon and others).

Paratyphoid Inoculation further modifies the clinical course. *The profuse rash* was never seen after T.A.B. vaccine.

Complications.—As in typhoid fever, but of far greater rarity. The incidence is further reduced by paratyphoid inoculation.

Diagnosis.—General diagnosis as in typhoid fever. Differentiation from typhoid fever and between paratyphoid A and B rests entirely on bacteriological and serological tests. The general diagnosis often depends on these tests.

ENTERIC FEVER IN INOCULATED PERSONS.

The clinical course of enteric fever, both typhoid and paratyphoid infections, is greatly modified during period of protection by previous inoculation. The condition is usually extremely mild, relapses are

unusual, and complications rare. Mortality about 1 per cent. The course may be of a few days' duration only, and all degrees exist, from slight transient malaise to, in very rare instances, typical enteric.

Symptoms frequently are pyrexia with persistent slow pulse, a furred tongue, and a doughy abdomen.

There is no doubt that many cases diagnosed during the War as 'enteric', on agglutination reactions, were really trench fever.

AGGLUTINATION REACTIONS IN PARATYPHOID FEVER.

Agglutination reactions in paratyphoid and also in typhoid fever are now known to be complicated by the occurrence of co-agglutinins.

Co-agglutinins or Group-agglutinins.—If an animal be inoculated with a certain bacillus, the blood serum may contain:—

1. SPECIFIC, PRIMARY, OR HOMOLOGOUS AGGLUTININS—viz., bodies which agglutinate the specific bacillus with which the animal was inoculated.
2. SECONDARY, HETEROLOGOUS, GROUP, OR CO-AGGLUTININS—viz., bodies which agglutinate bacilli of the same group as the specific bacillus.

Example.—If the specific bacillus used for inoculation be *B. typhosus*, the serum contains: (1) Specific agglutinins, which agglutinate *B. typhosus*; (2) Co-agglutinins, which agglutinate paratyphoid bacilli. The titre of the latter is usually much lower than the titre of the specific agglutinins.

The same phenomenon will occur in human beings with an attack of enteric fever or after inoculation with anti-typhoid vaccine.

'Absorption' of Agglutinins.—The agglutinins to any bacillus can be removed from a serum by Castellani's method of 'absorption'. The serum is saturated by the addition of a large number of the bacilli, incubated, allowed to stand for twenty-four hours, centrifuged, and the supernatant serum pipetted off. Result of absorption: (1) If the bacillus used for absorption be the specific bacillus, both the specific and the co-agglutinins are removed; (2) If the bacillus used be a heterologous bacillus, only the co-agglutinins for that bacillus are removed.

Example.—A serum prepared by inoculating an animal with *B. typhosus* contains both specific agglutinins to *B. typhosus* and co-agglutinins to *B. paratyphosus B*. (*a*) After absorption with the specific organism *B. typhosus*, serum agglutinates neither *B. typhosus* nor *B. paratyphosus B*. (*b*) After absorption with the heterologous organism *B. paratyphosus B*, serum does not agglutinate *B. paratyphosus B*, but still agglutinates *B. typhosus*.

Paratyphoid—'Absorption' of Agglutinins, *continued.*

THE METHOD CAN BE APPLIED:—

1. To ascertain which is the specific bacillus of a serum, e.g. in an attack of enteric.
2. To ascertain the identity or otherwise of two strains of bacilli which are both agglutinated by a serum.

Summary of Specific and Co-agglutinins in Human Enteric Infections.*

1. INFECTIONS WITH *B. Typhosus.*—Serum with cultures of:—
   B. typhosus.—Complete agglutination in 1–50. Often much higher.
   B. paratyphosus A.—Co-agglutinins absent or very slight.
   B. paratyphosus B.—Co-agglutinins common. Occasionally titre as high as to *B. typhosus.*
2. INFECTIONS WITH *B. Paratyphosus A.*—Serum with:—
   B. typhosus.—Co-agglutinins common. Titre may be as high as in *B. typhosus* infections (but usually for few days only).
   B. paratyphosus A.—Agglutinins tend to develop late, to be transient, and to be in low titre, rarely exceeding 1–40. A titre of 1–20 is proof of infection. May be absent, even in presence of co-agglutinins to *B. typhosus* and *B. paratyphosus B.*
   B. paratyphosus B.—As for *B. typhosus.*
3. INFECTIONS WITH *B. Paratyphosus B.*—Serum with:—
   B. typhosus.—Co-agglutinins common. Titre may be as high as in *B. typhosus* infections.
   B. paratyphosus A.—Co-agglutinins absent or very slight.
   B. paratyphosus B.—Complete agglutination in 1–50. Often much higher.

B. typhosus.—Complete agglutination in 1–50 is proof of an 'enteric' infection, but this *may* be due to a paratyphoid bacillus. Absolute proof of a *B. typhosus* infection (when required) depends on absorption, or isolation of bacilli.

B. paratyphosus A.—Comparatively inactive in production of agglutinins. Complete agglutination in 1–20 is proof of an *A* infection.

B. paratyphosus B.—A normal serum may agglutinate *B* in 1–50. Complete agglutination in 1–100 is proof of an 'enteric' infection, but this may be due to *B. paratyphosus A* or to *B. typhosus.*

AGGLUTINATION REACTIONS IN INOCULATED PERSONS.

After inoculation with typhoid vaccine, agglutinins to *B. typhosus* are present, and after T.A.B. vaccine, are present to the paratyphoid bacilli also. The titre and the duration of these agglutinins vary greatly. Agglutination reactions thus are greatly complicated.

* Titres apply only to microscopic methods.

The agglutinin-forming mechanism is in a highly sensitive condition (C. J. Martin), and liable to sudden activity even in health. Variations can only be accepted as evidence of enteric infection when the titres are very high, and they are rarely conclusive.

IDENTIFICATION OF ENTERIC, DYSENTERY, AND FOOD-POISONING BACILLI.

Three Groups.—(1) Enteric bacilli: *B. typhosus*, *B. paratyphosus A*, *B*, and *C*. (2) Dysentery bacilli: Shiga and Flexner strains. (3) Food-poisoning bacilli.

Methods of Differentiation.—(1) Cultural characteristics. (2) Agglutination with specific antisera. (3) Absorption of agglutinins in antisera (*see* AGGLUTINATION REACTIONS).

Summary of Methods of Identification.—

1. ENTERIC GROUP.—

Specific antisera will distinguish the four types from each other.

Cultural distinctions: *B. typhosus* produces no gas in carbohydrates. The paratyphoids produce gas in certain carbohydrates, but differ in action on milk, *A* causing permanent acidity, *B* and *C* a final alkalinity.

B. paratyphosus B has cultural characteristics identical with the food-poisoning group, and is agglutinated by *B. aertrycke* antisera. *Distinction necessitates absorption test. B. paratyphosus C* is not agglutinated by *B. paratyphosus B* antiserum.

Certain non-pathogenic bacilli closely resemble the paratyphoids, but have no action on dulcite and are not agglutinated by paratyphoid antisera.

2. DYSENTERY BACILLI.—Produce no gas in carbohydrates, and thus differ from paratyphoid and food-poisoning bacilli. Flexner and other strains distinguished from *B. typhosus* by agglutination with antisera.

3. FOOD-POISONING BACILLI (*see also* FOOD POISONING).—The cultural characteristics of the following bacilli are identical: (*a*) *B. enteritidis* (Gaertner). (*b*) *B. suipestifer*, the bacillus of hog cholera or swine fever (*B. aertrycke*). (*c*) *B. paratyphosus B* and *C*.

Gaertner's bacillus is readily distinguished from the others by agglutination with specific antisera.

The remaining members are all agglutinated to the same degree by antisera prepared for any one of them. Absorption of agglutinins (originally carried out by Bainbridge) gives the following results: *B. aertrycke* and *B. suipestifer* are the same organism; *B. paratyphosus B* is a different organism from these.

Typhoid—Identification of Bacilli, *continued.*

'Salmonella Group'.—The three bacilli, Gaertner, aertrycke, and paratyphosus B, have been grouped together as the 'Salmonella' or 'food-poisoning group.' The classification is unwise and erroneous: paratyphosus B does not cause an *outbreak* of 'food-poisoning' or of acute enteritis, although sporadic cases may resemble this; and it is not a 'food-poisoning' organism, reputed epidemics being due to lack of distinction from *B. aertrycke.*

The 'food-poisoning bacilli' are thus Gaertner and *B. aertrycke.* Of *B. aertrycke* several strains have already been recognized—e.g., Newport (Schültze), Mutton (Hutchens)—and the agglutinations with antisera, both before and after absorption, vary to some degree for the different strains.

CHAPTER II.

SEPTICÆMIA. PYÆMIA. TOXÆMIA.

Conditions in which a group of constitutional symptoms occur, with or without local manifestations of suppuration, due to the toxins of various micro organisms, usually of the common pyogenic bacteria.

Three Groups are recognizable:—

1. SEPTICÆMIA.—Characterized by presence and multiplication of organisms within the blood and by the absence of local abscess formation.
2. PYÆMIA.—Characterized by occurrence of multiple abscesses in the superficial tissues and internal organs
3. TOXÆMIA.—The organisms are confined to a focus, whence their toxins enter the circulation—e.g., in diphtheria. The 'sapræmia' of gynæcologists may be referred to this class: also the pyrexia and milder constitutional disturbances of simple suppuration.

The groups are artificial to a considerable extent, intermediate and unclassifiable conditions being common.

SEPTICÆMIA.

Etiology.—May arise from:—

1. LOCAL FOCI OF INFECTION.—Usually conditions without local formation of pus—e.g., post-mortem wounds—permitting the entry of organisms into the circulation. Also endocarditis.
2. 'CRYPTOGENIC' INFECTION.—Site or cause of entry not discoverable: subjects usually debilitated.

Bacteriology.—*Streptococci* most common. Numerous bacteria occur—e.g., pneumococcus, staphylococcus, gonococcus, anthrax, influenza, pyocyaneus, and bacilli of the coli-typhoid group.

Morbid Anatomy.—*Blood* often fluid and dark. *Spleen* large and soft. *Petechial hæmorrhages* common: especially on serous membranes. *Arterial walls* stained. *Kidneys* and other organs show cloudy swelling.

General Characteristics.—(1) *Rigors* and *sweats*. (2) *Pyrexia*: May be daily remissions or intermissions, or steady rise. (3) *Pulse*: Small, soft, and rapid. (4) *Gastro-intestinal disturbances*: Furred tongue, often dry; anorexia; constipation. (5) *Prostration* marked; rapid wasting. (6) *Mental symptoms*: Delirium if debilitated; may remain mentally clear. (7) *Pallor*. Conjunctivæ may be injected. Transient erythemata, etc., may occur. (8) *Hæmorrhages*, petechial or purpuric. (9) *Leucocytosis*: (*a*) Total leucocytes increased (10,000 to 20,000 per c.mm.); (*b*) Polynuclear cells relatively increased (up to 90 per cent or higher). (10) *Urine*: Albuminuria rarely absent.

In Very Acute Forms 'typhoid state' develops. Severe symptoms are: (1) *Skin* dry. (2) *Pulse* very small, soft, rapid, and 'running'. (3) *Temperature*: May either rise steadily or fall to subnormal. (4) *Prostration* extreme. *Delirium* usual. (5) *Diarrhœa and vomiting*. (6) Jaundice; hæmorrhages, hæmaturia. (7) *Blood*: Leucocytosis absent; may be definite leucopenia (1000 to 4000 cells per c.mm.) combined with high percentage of polynuclear cells.

PYÆMIA.

Etiology. *Focus of suppuration* present—e.g., septic wound, osteomyelitis, otitis media, appendicitis, septic arthritis. Spread of organisms due to septic emboli, thus: (*a*) From suppuration in portal system abscesses form in liver; (*b*) From external wounds, etc., suppuration spreads into general circulation.

Bacteriology.—Staphylococci and streptococci predominate. Other organisms rarely, as in septicæmia.

General Characteristics.—

1. GENERAL SYMPTOMS.—Resemble those of septicæmia. Sweats and rigors marked. Also superficial abscesses.
2. LOCAL SYMPTOMS, due to septic emboli, and local abscess formation. Especially: (*a*) In lungs—dyspnœa, cough, and hæmoptysis; (*b*) Pleurisy; (*c*) Pericarditis; (*d*) Spleen enlarged and painful; (*e*) Hæmaturia; (*f*) Cerebral abscesses.

Diagnosis.—

SPECIAL METHODS.—(1) Blood culture; (2) Blood count; (3) Agglutination for enteric group. Diagnosis often simple, with primary focus obvious.

Pyæmia, *continued.*

DIAGNOSIS FROM: (1) *Typhoid fever;* (2) *Infective endocarditis;* (3) *Malaria*—by examination of blood for protozoa and action of quinine; (4) *Acute miliary tuberculosis.* Occasionally: Impacted gall-stones; pyelitis; Hodgkin's disease (Pel-Ebstein type).

Treatment.—*Surgical treatment* if any indication present.

General treatment: Fluid freely; alcohol freely; fluid by intravenous, subcutaneous, or Murphy's drip method.

Vaccines, preferably autogenous. Antisera at present disappointing. Various drugs have been injected, and are under trial.

CHAPTER III.

ERYSIPELAS.

A spreading streptococcal inflammation of the deeper layers of the skin, with local and constitutional symptoms.

Etiology.—Commonest in *spring months.* Is contagious, conveyable by third persons or by bedding, etc., of a patient. *Onset* may be: (1) *Idiopathic*, commonly on face; (2) In puerperium. Also after surgical operations; from slight abrasions.

Alcohol, nephritis, diabetes, and debility are predisposing factors. *Recurrence* is common (especially facial).

Bacteriology.—A streptococcus originally described as a special strain, *Str. erysipelatis* (Fehleisen, 1884): now held to differ from *Str. pyogenes* only in lower virulence. Note, however: (1) Erysipelas is transmitted as such from one patient to another; (2) Purulent streptococcal foci do not lead to erysipelas.

Morbid Anatomy.—*Streptococci* are present in the spreading edge, in the lymphatics of the skin and subcutaneous tissues.

Symptoms (facial erysipelas).—

ONSET.—Malaise, rigor, pyrexia. Commences over nose and cheeks or at local abrasion.

LOCAL SYMPTOMS.—(1) Skin red, hot, smooth, tense, and œdematous; (2) Blebs common; (3) Definite spreading red edge develops; (4) Advances at the edge, while centre fades. Face and features swell enormously, especially eyes, lips, and scalp. Neck swollen and glands enlarged. Pus may form under scalp. Mouth, throat, and larynx may be involved.

CONSTITUTIONAL SYMPTOMS.—*Temperature* high: usually no remissions. Symptoms severe in old, alcoholic, or debilitated subjects. *Delirium*, especially in alcoholics or when scalp is involved. Albuminuria usual.

Complications.—Œdema of glottis serious. Meningitis rare, even when meningeal symptoms are present. Rarely: pneumonia, pyæmia, septicæmia.

Course and Prognosis.—Self-limited. Spreading edge dies out. Temperature often falls about fourth to fifth day. Mortality very low if previous health good.

Treatment.—

ISOLATION AND DISINFECTION NECESSARY.

GENERAL TREATMENT.—Light diet. Much fluid. Brisk purge. Alcohol freely. No incisions, unless pus formation

LOCAL TREATMENT.—Ichthyol ointment (1-4 lanolin) with lint mask. In mild cases, cooling applications sufficient: cold water or evaporating lead and opium lotion. Tincture of iodine may be painted on skin, ½ to 1 inch from spreading edge (to promote leucocytosis).

DRUGS.—Tinct. ferri. perchlor. ʒss to ʒj, four-hourly: often recommended.

FOR HYPERPYREXIA.—Antipyretics (phenacetin, etc.); or, if necessary, bathing, etc., as in enteric.

IN CONVALESCENCE.—Tonics and fresh air necessary.

VACCINES AND ANTISERA.—Not of proved effect.

CHAPTER IV.

DIPHTHERIA.

A specific infectious disease due to the Klebs-Loeffler bacillus, and characterized by local symptoms due to a fibrinous exudate, usually on mucous membranes of fauces or larynx, and by constitutional symptoms due to toxins produced by the bacilli at the site of exudate.

Etiology.—

GEOGRAPHICAL DISTRIBUTION.—Almost universal, but most prevalent in temperate and cold climates. *Endemic* in all large towns; epidemics not infrequent.

SEASON.—Especially in last quarter of year. Highest in dry years. In England, slight fall in August, and maximum in October and November.

AGE.—Extremely important. Frequency and mortality are greatest between 1 and 5 years: period includes nearly 80 per cent of deaths. Over 10 years, frequency less and mortality lower. Not frequent under 6 months (inherited immunity).

SEX.—Slightly commoner in girls, from frequent kissing.

INDIVIDUAL SUSCEPTIBILITY appears to be important.

Modes of Infection.—Very contagious. Transmission usually occurs almost directly from one person to another—e.g., from

Diphtheria—Modes of Infection, *continued.*

kissing, by interchange of pencils, etc., at schools. Sources of infection :—

1. DIRECTLY FROM INDIVIDUAL with typical active diphtheria.
2. INFECTED ARTICLES.—Bacilli may live for months. Also conveyance of bacilli by 'third persons.'
3. DIPHTHERIA CARRIERS—i.e., bacilli present in throat, but no clinical symptoms.—(*a*) *Healthy contacts* who have never shown symptoms of an attack; (*b*) *Patients who have recovered* but still carry bacilli in the throat.
4. SUBJECTS OF ATYPICAL DIPHTHERIA—e.g., mild tonsillitis. Severe attack may occur in infected individual.

In the following the human contact is not so direct :—

5. EPIDEMICS DUE TO MILK. Established in several outbreaks. Cows may carry virulent diphtheria bacilli on their udders, though they are not found elsewhere; possibly an ulcer is infected by a human carrier. Occasionally diphtheria carriers may infect milk. (*Note.*—Non-pathogenic diphtheroid bacilli are often present in milk and cheese.)
6. ACCIDENTAL INFECTION FROM CULTURES.
7. ANIMALS.—Cats can convey infection.

No transmission takes place by water or by air—viz., sewer gas, drains, etc.—as formerly believed.

One attack does not confer immunity.

Bacteriology.—*B. diphtheriæ* was discovered by Klebs in 1883, and isolated by Loeffler in 1884.

MORPHOLOGICAL CHARACTERS.—A non-motile, non-sporing bacillus. Length and appearance very variable: varies from a short bacillus with rounded ends, to irregular forms with swollen 'clubbed' extremities; the latter 'involution' forms are common in cultures of more than forty-eight hours' growth. May stain uniformly, but more commonly shows 'beaded' appearance or irregular staining. The arrangement of the bacilli in films from cultures is often characteristic, the groups resembling 'Chinese letters,' due to the organism bending lengthways before division. From tissues, bacilli are often single, unless numerous.

STAINS.—*Gram-positive*, but fairly easily decolorized. Better stained as routine by Loeffler's alkaline methylene blue, or by toluidin blue. Neisser's stains, the original Bismark-brown or the cresoidin method, exhibit the granules better, but are preferably used only as confirmatory in doubtful cases, and not as initial routine methods.

SPECIAL CHARACTERISTICS.—(1) Irregular staining; (2) Arrangement.

CULTURAL CHARACTERS.—Grows well on all ordinary media in subcultures. Initial cultures from tissues to be made on Loeffler's blood-serum. *Growth is rapid at 37° C.* Colonies may be visible in twelve hours: bacilli may be found in films after six to eight hours. Very resistant to drying.

DISTRIBUTION OF THE BACILLUS IN THE TISSUES.—

1. IN THE MEMBRANE.—Mainly in superficial portions and on surface. Bacilli do not penetrate below membrane.
2. IN OTHER SITES, especially mucous membranes.—Occasionally present in *rhinitis, conjunctivitis*, and, less commonly, otitis media; also in vulva. Rarely in wounds, and very rarely in ulcerative endocarditis.

INOCULATION INTO ANIMALS.—Subcutaneous inoculation into leg of guinea-pig with forty-eight-hour broth culture or suspension is used to test virulence of bacilli. *Result*: Death in thirty-six to seventy-two hours, with rapid loss of weight, great œdema at site of inoculation; *hæmorrhages into suprarenals* and serous membranes, bacilli at site of inoculation only.

TOXINS OF THE KLEBS-LOEFFLER BACILLUS.—Roux and Yersin isolated toxins from cultures of the bacillus which on inoculation caused symptoms of the disease except for absence of membrane. They proved that death in diphtheria is due to action of toxin and not to extension of bacillus. Animals can be immunized to a high degree by injections of the toxin.

BACTERIA ASSOCIATED WITH THE DIPHTHERIA BACILLUS.—*Str. pyogenes* is most important, and is the usual cause of suppuration of the glands, and occasionally leads to general septicæmia.

Diphtheroid Bacilli.—

DIFFICULTY OF DISTINGUISHING THE KLEBS-LOEFFLER BACILLUS FROM DIPHTHEROID BACILLI.—Caused by: (1) Presence of the bacillus in atypical clinical conditions; (2) Existence of closely similar non-virulent bacilli; and (3) Production of membranes by other organisms.

1. TRUE DIPHTHERIA BACILLI PRESENT, BUT CLINICALLY ATYPICAL.—

   The bacilli may be found in conditions with clinical appearance of simple tonsillitis or angina without membrane. Cardiac failure or peripheral neuritis may follow, or virulent symptoms occur in persons subsequently affected.

   The bacilli may occur in healthy throats, i.e., 'diphtheria carriers.'

2. PRESENCE OF NON-VIRULENT BACILLI RESEMBLING KLEBS-LOEFFLER.—

   Hofmann's Bacillus or Pseudo-diphtheria Bacillus.—May be present in various anginal and tonsillitic conditions; possibly infective, but sequelæ of diphtheria are absent. Occurs in healthy throats.

   B. Xerosis and Skin Diphtheroid Bacillus.—Frequently present on conjunctiva even in health (is not the cause of xerosis). Closely resembles Klebs-Loeffler bacillus. Similar organisms are frequent on skin and in wounds. Are non-virulent, giving negative inoculation results on animals.

Diphtheria—Diphtheroid Bacilli, *continued.*

3. DIPHTHEROID INFLAMMATIONS.—Membranous inflammations occur in children, especially in acute specific fevers—most often in scarlet fever, less so in measles. Organism may be streptococcus, or diphtheria or diphtheroid bacillus. Streptococcal membranes are true membranes, and separate without leaving a bleeding surface.

HOFMANN'S BACILLUS—CHARACTERISTICS.—Short, plump bacillus with round ends. Involution forms and polar staining and 'beading' absent. Arrangement in cultures as Klebs-Loeffler bacillus, but frequently more definitely parallel. Gram-positive. Grows more profusely than, and has slight cultural differences from, true diphtheria bacillus—e.g., does not ferment dextrose but these are insufficient for differentiation. *Fatalities and complications* do not occur. *Is non-pathogenic to animals.* Is a separate organism, and not a modified diphtheria bacillus.

DIFFICULTIES ARISING FROM DIPHTHEROID ORGANISMS.—In great majority of cases, no doubt arises. Use Neisser's stain when in doubt. Difficulty is caused by short non-involuted form of Klebs-Loeffler bacillus, which closely resembles Hofmann's and may be but slightly virulent to animals.

Greatest difficulty is the urgent need for a rapid opinion. In general: (1) A bacillus isolated from the throat with the morphological and staining reactions of diphtheria should be accepted as true diphtheria. (2) A similar bacillus isolated from elsewhere in the body should not be accepted as diphtheria until virulence proved to animals (Muir and Ritchie). (3) Cases of Hofmann's bacillus from the throat, in presence of symptoms, should be isolated, with precautions the same as, but not greater than, in a severe tonsillitis. When necessary it may be referred to as 'infective tonsillitis'. Antitoxin not necessary.

Schick Test.—

THEORY OF TEST.—(1) Presence of $\frac{1}{50}$ unit of antitoxin per c.c. of blood gives immunity to diphtheria; often present in normal persons. (2) Such amount prevents any reaction after injection of $\frac{1}{50}$ M.L.D. (minimal lethal dose) of diphtheria toxin.

TECHNIQUE.—Inject *intradermally,* not subcutaneously, 0·2 c.c. of a saline solution containing $\frac{1}{50}$ M.L.D. (obtainable from Burroughs Wellcome & Co. and others). The flexor surface of the forearm is convenient. In opposite arm inject, as control, toxin heated to 75°C for 10 minutes.

REACTIONS.—

1. POSITIVE.—Sharply circumscribed area of redness, diameter ½ to 1 in.; appears in 24 hours, maximum in 72 to 96 hours; duration 7 days, pigmentation up to 10 days.
2. NEGATIVE.
3. PSEUDO-REACTION.—Ascribed to proteins of toxin, but doubtful. Commonest in adults; commences at 5 to 10 years; rare at younger ages. A larger and less circumscribed area of brighter red, appearing in 18 to 24 hours,

maximum in 20 to 30 hours, and fading in 3 days. Hence necessity of control.

4. COMBINED PSEUDO- AND POSITIVE REACTIONS.
Pseudo-reactions are distinguished specially by shorter duration.

RESULTS AT VARIOUS AGES.—Under 6 months, all negative (inherited antitoxin); from 6 months to 6 years, 50 to 70 per cent positive; percentage falls to 20 per cent in adults.

INTERPRETATION OF RESULTS.—

a. NEGATIVE.—Indicates immunity. Such contacts do not require antitoxin.

b. POSITIVE.—Indicates susceptibility. Such contacts require antitoxin or active immunization.

c. PSEUDO-REACTIONS.—Negligible.

Active Immunization: Antitoxin-Toxin Mixture.—Mixtures in use vary: Burroughs & Wellcome's contains 3 L + doses of toxin and 3·5 units of antitoxin in 1 c.c. (An L + dose of toxin is the amount which, mixed with 1 unit antitoxin, kills a 250-grm. guinea-pig at end of 4 days.)

METHOD.—Inject subcutaneously for a child at weekly intervals 0·25, 0·5, and 1 c.c. Considerable local reaction is common, but constitutional symptoms are rare. Immunity develops in 3 weeks to 3 months, and is known to last 1½ years.

Carriers.—Presence of bacilli without clinical symptoms occurs in: (*a*) 'Healthy' or 'contact carriers'; (*b*) 'Convalescent carriers', recovering from an attack. Virulence of such bacilli to animals varies greatly; often avirulent.

Certain authorities believe:—

1. Bacilli from most healthy carriers are non-virulent.
2. Non-virulent bacilli never regain virulence.
3. Non-virulent bacilli cannot cause diphtheria.
4. Therefore non-virulent bacilli are not a menace.
5. No legal (in Great Britain) or practical reason exists for notifying a healthy carrier or for removal to a fever hospital.

Note.—Above statements admittedly need further research. Practitioners at present obviously incur responsibility if regarding carriers lightly.

Morbid Anatomy.—Characteristic change is membrane formation in upper air-passages. The membrane is produced by changes in the superficial layers of the tissues, and is thus a '*false membrane*'. Its formation is due to action of toxins of the diphtheria bacilli.

DIPHTHERITIC MEMBRANE.—

COMMON SITES.—*Tonsils and neighbourhood*, and *larynx*. Also occurs on pharynx, trachea, epiglottis, nares. In fatal cases, often in accessory sinuses. Rarely, on conjunctiva.

MACROSCOPIC CHARACTERS.—

1. *Colour of membrane* grayish-white; later may darken.
2. *Adherent, and leaves bleeding surface on separation*. In later stages separates easily.

Diphtheria –Morbid Anatomy, *continued*

3. *Is superficial* only in rare cases extends deeply. Disappears by disintegration.

HISTOLOGY Membrane is formed by coagulative necrosis of epithelial cells, with exudation of fibrin and, in deeper layers, of polynuclear cells Frequently the epithelial cells are shed early *Tissues below membrane are but little affected Diphtheria bacilli present* mainly on surface and in superficial layers do not penetrate deeply

FAUCIAL DIPHTHERIA - Initial slight catarrh of fauces Membrane formation commences usually at one spot either on tonsils or at junction of uvula and tonsil, and spreads over tonsil pillar of fauces uvula, over soft palate, and often over pharynx, *is not confined to tonsil*

LARYNGEAL DIPHTHERIA Membrane may include nasal Spreads upwards to epiglottis downwards may extend even to bronchioles Faucial membrane usually present

LYMPHATIC GLANDS *Enlarged in neck and under jaw* in severe cases extreme Mainly due to secondary streptococcic infection, and not rapidly affected by antitoxin.

HEART —Myocardial changes important Fatty degeneration often marked Endocarditis, very rare

PULMONARY LESIONS *Bronchitis and bronchopneumonia* common and fatal, especially in laryngeal type Pneumococcus is commonest organism Klebs Loeffler bacillus rare *Membrane* may extend down trachea to bronchi rarely to bronchioles

NERVOUS SYSTEM Parenchymatous degeneration of peripheral nerves, sensory and motor, in diphtheritic paralysis

OTHER CHANGES NOT CHARACTERISTIC

BLOOD Definite leucocytosis, and relative increase of polynuclear cells

KIDNEY Fatty degeneration, and rarely nephritis

LIVER AND SPLEEN Toxic changes

Symptoms.—

INCUBATION PERIOD Usually two to five days, most commonly two. Rarely, bacilli may lie latent for prolonged period before symptoms arise

EARLY SYMPTOMS - General malaise. Temperature about 101° rarely exceeds 103° Slight hoarseness · sore throat often unnoticed in children. Face gray. May be convulsions in infants Knee-jerks often absent Trace of albumin very frequent

CLINICAL TYPES —(*A*) Faucial, (*B*) Laryngeal, (*C*) Nasal

A. FAUCIAL DIPHTHERIA.- In children is a silent disease—little pain, complaint, or crying symptoms being toxæmic.

Early Symptoms.- As above. Some difficulty in swallowing. Tonsils · general catarrh; membrane often commencing on first day. Glands in neck and under jaw tender and slightly enlarged on affected side

Third Day.—*Membrane* on tonsils, palate, and uvula: may fill aperture. Glands larger. Temperature is

variable General malaise and toxæmia Pain as a rule only on swallowing

Fourth to Fifth Day Membrane extensive Glands large Breath very heavy Tongue furred Urine reduced Albumin almost constant

Favourable Cases Subsequently membrane disintegrates Signs disappear Convalescence in seven to ten days Constitutional symptoms generally definitely in proportion to extent of membrane

Severe Cases Waxy face Pulse feeble, rapid, or often low the latter very serious Temperature may be high or low Membrane usually extensive Nasal discharge common Vomiting Albumin increases Prostration marked *Death* from cardiac failure often sudden usually in three to eight days *Larynx* often involved

Tonsils may show following variations (1) Pain late exudate is in follicular tonsillitis (2) General pultaceous exudate, (3) Miliary membrane at several points (4) Catarrh In severe cases with little membrane virulent bacilli often numerous in nares

LARYNGEAL DIPHTHERIA Commonest about three years of age Nearly always secondary to faucial diphtheria, and faucial membrane cervical glands, and symptoms present

Early Stage An acute laryngitis producing croup', viz (1) Hoarseness (2) Harsh cough (3) Inspiratory stridor (4) Inspiratory recession above clavicle

Clinical Varieties

1 Onset sudden but symptoms not severe. Paroxysm of dyspnœa for few hours due to spasm of glottis Membrane slight Prognosis good

2 Onset less sudden Dyspnœa become continuously worse without spasms Colour livid Cyanosis and croup' increase Restlessness vomiting and coma Condition associated with spread of membrane down trachea Pulmonary complications common Prognosis very bad

Temperature rarely high unless faucial symptoms marked

In adults, laryngeal diphtheria is rare, but is often overlooked, width of larynx prevents blockage, and hence there is no croup Membrane spreads to fine bronchi, with severe symptoms and high mortality

C NASAL DIPHTHERIA Occurs in two forms

1 *Primary Membranous Rhinitis*—Nasal discharge Membrane often very extensive Symptoms often slight, and cause overlooked

2 *In Faucial Diphtheria*—Discharge may be hæmorrhagic Symptoms usually severe though membrane slight.

Complications.—

1 PULMONARY.—*Bronchitis and bronchopneumonia* nearly always present in severe cases.

2 CARDIAC—*Irregularity* very common faint murmur frequent.

Diphtheria –Complications, *continued.*

Marked irregularity, and especially slow pulse, of serious prognosis: often sudden death. Severe cardiac symptoms not common in acute stage.

3. ALBUMINURIA.—*Almost constant*, and *very early*, not uncommon on first day. Amount large in severe cases. Anuria serious. Subsequent nephritis very rare.
4. VOMITING.—Dangerous sign.
5. RASHES.—Diffuse erythema occasionally even in absence of antitoxin.

Sequelæ. Of extreme importance: (*A*) Paralysis; (*B*) Cardiac failure.

A. POST-DIPHTHERITIC PARALYSIS. Strict sequel: occurs in second or third week of convalescence: of toxic origin.

FREQUENCY.—10 to 15 per cent: higher in adults. Most common *in faucial type. Usually following severe cases*, but also in mild forms.

EFFECTS OF ANTITOXIN TREATMENT (Goodall). Total frequency not diminished, but paralysis of less severity ascribed to survival of great numbers of severe cases. Paralysis rare when antitoxin given on first or second day.

PROGRESS.—From onset of paralysis takes two to seven weeks to become complete. Progress may be arrested at any stage.

ORDER OF PROGRESSION.—(1) Palate; (2) Eye; (3) Limbs, occasionally (4) Trunk; (5) Diaphragm; (6) Intercostals. *Special senses never affected.* Facial paralysis rare. Involvement of sphincters very rare.

1. *Palate.—Nearly always affected first.* Earliest signs: *Nasal voice; regurgitation of food through the nose.* On examination: *Palate relaxed, motionless, insensitive, and reflex absent*: changes often incomplete in milder degrees. *Constrictor of pharynx* affected in severe cases, whence difficulty in deglutition, and choking. *Larynx* affected in late stages with widespread paralysis: paralysis of adductors, causing hoarseness and weak cough: may simulate relapse of laryngeal diphtheria. Anæsthesia of larynx may lead to aspiration of food.
2. *Eye.*—Frequency of affection next to palate. Most common is *loss of power of accommodation* from paralysis of ciliary muscles, revealed by difficulty in reading. *External rectus* most commonly affected of extrinsic muscles. Diplopia and squint of every grade to complete opthalmoplegia externa (very rare). *Pupils* often sluggish: may react to light and not to accommodation (very rare apart from diphtheria). Argyll Robertson pupil very rarely.
3. *Limbs.—Legs* more frequently affected than arms; commences with weakness in walking. *Knee-jerk*, and deep reflexes abolished. With complete

paralysis, *wasting* of muscles is often extreme. *Sensation* is usually affected, but marked loss is unusual. Reaction of degeneration very rare.

4. *Trunk Muscles.*—May be inability to move head.
5. *Diaphragm.*—Special danger to lungs from accumulation of mucus.
6. *Intercostals.*—Respiration seriously affected.

A generalized type of paralysis occurs in which the last three groups of muscles are specially affected. otherwise their involvement is uncommon.

COMMON COMBINATIONS of paralyses are: (1) Palate only or ocular only. (2) Palate and slight ocular, especially accommodation. (3) Palate, slight ocular, knee-jerks absent, and [illegible] weakness of legs. These three forms are frequent; recovery is usual in two to three weeks. (4) Severe: palate, pharynx, eyes, and legs. (5) Generalized form palate and eyes slight, trunk and limbs marked.

CAUSE OF DEATH IN PARALYSIS.—(1) *Respiratory failure* from paralysis of muscles; aspiration pneumonia; massive collapse of lungs. (2) *Cardiac failure.*

PROGNOSIS IN PARALYSIS.—When mild, recovery complete in a few weeks. Severe cases, prolonged. *Paralysis never persists with life.* Mortality in adults very low.

B. CARDIAC FAILURE.—Apart from acute stage, failure most common in third week. Cardiac symptoms may occur as follows:—

1. Patient with paralysis of any degree allowed to get up: may be suddenly fatal.
2. Patient without paralysis allowed up under three weeks after severe attack.
3. Rarely occurs in bed, after severe attack, on slight exertion.

Slight symptom is tachycardia.

Serious symptoms are severe precordial pain, vomiting, irregularity, and dilatation: mortality very high

Diagnosis.—

1. BACTERIOLOGICAL METHODS.—Rub sterile swab on membrane or tonsil; inoculate blood-serum; incubate twelve hours at 37° C.; a preliminary examination may be made in eight hours. The swab is also rubbed directly on a microscope slide, and the smear stained and examined: positive results not uncommon, but negative results of little value. Presence of Klebs-Loeffler bacilli is absolute proof: absence in cultures, with definite membrane present, is negative proof. With suspected laryngeal diphtheria, repeat examination if negative.

A negative examination may result *erroneously* from: (*a*) Use of antiseptics on fauces: should not be employed for four hours previously. (*b*) Membrane not touched by swab. (*c*) Mixed infection: careful examination of film necessary.

Severity of attack cannot be judged from culture; but with pure cultures it is usually severe.

Diphtheria—Diagnosis, *continued.*

For difficulties of bacteriology, *see* BACTERIOLOGY. Inoculation into animals in doubtful cases.

Never wait for bacteriological report before commencing treatment.

2. CLINICAL DIAGNOSIS. Early albuminuria and absence of knee-jerks are often suggestive.

a. FAUCIAL DIPHTHERIA.—Diagnosis necessary from: (i) Follicular tonsillitis; (ii) Scarlet fever. Less commonly, from secondary syphilis, thrush fungus, quinsy, Vincent's angina, and herpes of palate. Scalds of pharynx and curds of milk have caused mistakes.

Follicular Tonsillitis.—Onset rapid. *Temperature* high, 104°. Face flushed. Any membrane present is limited to tonsils, and leaves no bleeding surface on separation.

Scarlet Fever.—Sudden onset with vomiting. *Temperature* high, 103°. *Pulse* rapid. Face flushed: circumoral pallor. *Tongue* strawberry. *Rash*: punctate erythema.

Quinsy.—Diphtheria never suppurates.

b. LARYNGEAL DIPHTHERIA.—Diagnosis from: (i) Acute laryngitis; (ii) Measles; (iii) Retropharyngeal abscess, (iv) Bronchopneumonia. Less commonly from laryngismus stridulus, foreign body, and papilloma of larynx.

Acute Laryngitis.—Often difficult. Constitutional symptoms slight. Bacteriology. Primary acute laryngitis in infants is nearly always diphtheria.

Measles. Catarrhal symptoms. Koplik's spots. No membrane present. Later, typical rash.

Retropharyngeal Abscess.—Recognized by palpation.

Bronchopneumonia.—Expiratory stridor. Retraction of lower ribs.

Laryngismus Stridulus.—Recurrent nocturnal attacks of dyspnœa. Sudden onset. No membrane. Slight general symptoms. Spasm relieved by warm bath or by chloroform.

Papilloma of Larynx.—Hæmorrhage occurs.

Association with Other Specific Fevers. Frequent with measles and scarlet fever (q.v.); prognosis serious.

Prognosis.—

INJECTION OF ANTITOXIN.—Prognosis varies almost directly with day of injection: mortality under 2 per cent when given on 1st or 2nd day: with recent larger doses is practically nil in faucial forms. Death-rate rises rapidly with delay, about 5 per cent when given on 3rd day, and 10 per cent when given on 4th day.

LARYNGEAL FORM.—Death-rate much higher than faucial form, but very low if injection given on 1st day.

AGE.—Mortality decreases rapidly after 7 years. The younger the age, the higher the mortality.

DANGEROUS SYMPTOMS.—Very irregular *pulse*, especially if slow. Low temperature with symptoms of prostration. Repeated vomiting. Marked albuminuria. Convulsions.

In Faucial Diphtheria. Extensive membrane. Great enlargement of glands.

In Laryngeal Diphtheria. Marked obstruction. Pulmonary symptoms.

In Nasal Diphtheria. Free hæmorrhage.

In Paralysis.—Extensive paralysis. Involvement of respiratory muscles. Signs of cardiac weakness. Vomiting.

Prophylaxis.—The following measures should be adopted:—

Complete isolation of patient, disinfection of clothes, etc.

Patient not discharged until Klebs-Loeffler bacilli absent: three examinations at intervals of at least four days, preferably commencing on twenty-first day. (Many competent authorities consider these examinations unnecessary if patient be clinically free from all symptoms.)

Examine throats of contacts bacteriologically. *See also* Schick Test.

Prophylactic doses of antitoxin should only be given for a definite reason—e.g., surgeon after performing tracheotomy. Often given to contacts in institutions, etc.: apart from short duration of protection and occurrence of after-effects, note that the results of such injections are not fully known, and may possibly lead to 'carriers' without development of symptoms.

All attendants should wear gowns and caps, and gauze masks over nose and mouth; pay special attention to sterilization of hands, gargle with weak carbolic lotion or antiseptics.

Treatment.—Methods of primary importance are: (1) Injection of antitoxin; (2) Rest. Of less importance are general hygiene, diet, local treatment, treatment of special symptoms.

1. INJECTION OF ANTITOXIN.—

Dosage.—Varies greatly with day of disease, and also with severity and clinical type. When in doubt, give a large dose. The general aim is to give all the antitoxin necessary within at most 24 hours, and not spread it over several days.

Seen 1st day of disease: Give 4000 to 8000 units, depending on age and severity, repeat in 8 to 12 hours. In laryngeal type, inject 6000 to 8000 units: repeat in 8 to 12 hours: when very severe, 10,000 units, and repeat twice within 24 hours. On 2nd day, repetition to be judged by condition; in faucial type, frequently unnecessary; in laryngeal type, advisable: single dose of amount as above. On subsequent days, depending on condition.

First seen after 1st day: Dosage increased by one-half for each day.

Children require a dose almost similar to adults: under 2 years, give two-thirds of above.

Desired result is general improvement and shrivelling of membrane, commencing in 12 to 24 hours.

Method of Administration.—

Subcutaneous injection into flank, etc., advisable as routine method. Use carefully sterilized syringe and needle: sterilize skin with iodine. Plunge needle well through

Diphtheria—Treatment, *continued.*

skin. Injection must be subcutaneous, and not into skin. Only freshly-opened phial of serum to be used.

Intravenous injection.—Under the conditions existing in experiments, results prove that this is most efficient method: from clinical results, evidence is less definite. It should be reserved for cases of great urgency. In infants, technical difficulties are considerable. Inadvisable if patient has previously received serum; anaphylaxis may be serious. Always give *subcutaneously* at once in preference to waiting for intravenous injection. Dosage: about two-thirds of amounts given above.

Oral and *Rectal* administration valueless.

AFTER-EFFECTS OF ANTITOXIN.—

a. SERUM RASH.—*Onset* seven to fourteen days after injection—usually ten days. *Urticaria* or *erythema*: may closely resemble measles. *Bathe* with lead lotion. The irritation is so extreme that morphia is frequently necessary. Calcium lactate has no preventive effect. Pyrexia and joint pains not uncommon.

b. ANAPHYLAXIS OR HYPERSENSITIVENESS.—Occurs in those who have had previous serum injection more than ten days previously: may be many years. Symptoms may develop with great rapidity, especially with intravenous injections; more commonly in half an hour to three hours; occasionally in one or more days. In acute cases, rapid onset of collapse. When less severe, shivering or rigor, dyspnœa, cyanosis, vomiting, varying degree of cardiac weakness and prostration, rash. Very rarely fatal except in asthmatics. *The possibility of anaphylaxis is never a contra-indication to subcutaneous injections for curative purposes.*

Desensitization.—*If intravenous injections* are considered essential, preliminary desensitization may be practised. Inject at intervals of five minutes successively 0·5 c.c., 1 c.c., 2 c.c., 5 c.c. of serum. If no symptoms occur, continue with complete dose. If symptoms occur, wait for half an hour before next injection, or give dose subcutaneously.

Treatment of Symptoms.—Stimulants. *Adrenalin* 1–1000, 3 to 5 minims hypodermically; if urgent, 1–5000, 3 to 5 minims intravenously, is recommended.

2. REST IN BED.—*Must be absolute, lying flat.*

DURATION.—In mild cases, for three weeks after membrane disappears. When severe, for at least three weeks after disappearance of symptoms, and period increased at slightest indication.

Each stage in getting up and convalescence should be extremely gradual, and pulse watched. Thus, for several days patients

should be sitting up in bed. Risk of cardiac failure is present from onset, and persists into convalescence.

GENERAL HYGIENE.—Remove carpets, etc. Temperature of room 63°. Free ventilation. Air not too dry, especially in laryngeal type (use bronchitis kettle). Give calomel.

DIET.—Milk. Custard and semi-solids in older children. If vomiting, peptonize milk: stomach wash rarely possible.

LOCAL TREATMENT.—Aims at cleanliness. Does not kill bacilli: omit if causes struggling. Syringe fauces and nares (if discharge): use warm water or salt and water. If less severe, swab with 1 per cent carbolic. For nasal form or septic discharge, syringing essential (listerine and borax); for profuse hæmorrhage, syringe with ice-water.

TREATMENT OF SPECIAL SYMPTOMS.—

COLLAPSE AND CARDIAC FAILURE. Brandy. Injections of caffeine sodium salicylate, or camphor.

PARALYSIS.—*Rest in bed*, absolute and prolonged. Give liberal diet, and arsenic and strychnine. *For severe regurgitation of food*: nasal tube in infants, stomach tube in adults. *Paralysis of respiratory muscles*: raise foot of bed; oxygen. *Wasting of muscles*: massage, electricity.

LARYNGEAL OBSTRUCTION. *Indications for tracheotomy*: *increasing dyspnœa*, inspiratory recession above clavicles, and restlessness. *Intubation* only in hospitals.

'DIPHTHERIA CARRIERS'.—Syringe nose, and also fauces: use antiseptics. Vaccines have been disappointing.

CHAPTER V.

THE PNEUMONIAS.

LOBAR PNEUMONIA.

(*Croupous Pneumonia.*)

An acute specific disease caused by the pneumococcus and characterized by toxæmia, consolidation of the lungs, and a fever which usually ends by crisis.

ETIOLOGY.

FREQUENCY.—Accounts for 5 to 10 per cent of all deaths.

AGE.—Frequency increases to 6th year, falls to the 15th year, and then again increases, especially for later decades.

SEX.—Males 2 or 3 to 1 female: probably due to conditions of life. Incidence equal when in similar conditions, e.g., prisons.

GEOGRAPHICAL DISTRIBUTION.—Universal: somewhat less frequent in tropics.

RACE.—Negroes and coloured races have high incidence and mortality when placed under abnormal conditions.

Lobar Pneumonia—Etiology, *continued*

EPIDEMICS —Outbreaks may affect households, institutions, and wider areas (Most of the recorded epidemics are insufficiently studied The possibility of 'influenza' especially affects epidemics)

Factors Increasing Liability to Attack. -

1 SEASON —Incidence highest in winter and spring.
2 OCCUPATION —Outdoor occupations show a higher incidence
3 PREVIOUS ATTACK —Frequently several attacks occur One attack probably predisposes to a second
4 COLD —Pneumonia frequently follows exposure
5 DEBILITY due to any cause
6 ALCOHOL is a specially important factor in prognosis
7 OTHER DISEASES Some diseases especially predispose to pneumonia, e g , influenza, and chronic debilitating conditions
8 TRAUMA —Attack may apparently follow directly upon injury particularly of chest not necessarily any lesion of the lung

As the pneumococcus is frequently present in the fauces of healthy persons, these factors are supposed to act by reducing the resistance of the body to its effects

BACTERIOLOGY.

The Pneumococcus.—

MORPHOLOGY —Typically a lance shaped coccus occurring in pairs —i e , a diplococcus In body fluids has a *capsule* which is recognizable but unstained except by special methods capsule lost in cultures May occur in short chains of 4 to 8 cocci when grown in fluid media *Gram-positive* In body fluids is extracellular, phagocytosis not occurring Cultures are necessary before the coccus can be identified with certainty

CULTURAL CHARACTERS Most important are Fine colonies on agar, no growth on gelatin, acidifies raffinose broth and inulin, usually *coagulates milk* Thus differentiated from streptococci and staphylococci Growth is very delicate, and cultures usually die out in a few days

SYNONYMS *Micrococcus lanceolatus, Diplococcus pneumoniæ*

Other Organisms present in Lobar Pneumonia.— *Str* and *Staph pyogenes,* Friedländer's pneumobacillus, *B. influenzæ,* and rarely *B diphtheriæ, B typhosus,* and other bacteria, may be associated with pneumococcus in lobar pneumonia

B. Pneumoniæ of Friedlander.— A short non-motile bacillus with rounded ends In tissues often as a diplobacillus and with capsule. Gram-negative. Produces acid and gas in dextrose, lactose, mannite, and maltose. Belongs to the colon group of bacilli. Is never the cause of true lobar pneumonia. (Described by Friedländer in 1883 as the cause of pneumonia)

Immunity. Specific Therapy. Prophylactic Inoculation. —Animals can be immunized by injections of pneumococci, attenuated by heat or other methods Duration of immunity

is a few weeks Serum of immunized animals is protective to some extent against injections with pneumococci also contains agglutinins

STRAINS OF PNEUMOCOCCI The Rockefeller Institute by agglutination experiments with sera prepared is above separated four types Relative frequency Type I 33 per cent mortality 25 per cent Type II 29 per cent mortality 30 per cent Type III 13 per cent mortality 45 per cent Type IV 20 per cent, mortality 16 per cent The remaining 5 per cent is composed of a few abnormal strains Type III slightly differs culturally (*Pneumococcus mucosus*)

PNEUMOCOCCIC ANTISERA — Antisera have been prepared for each group except that Type IV includes various strains and there is no group antiserum Only Type I antiserum is of value in treatment reduces Type I mortality to 7 5 per cent no effect on other types Rules for administration (1) Administer only to proved Type I cases Do not give polyvalent sera or to other types (2) Inject intravenously 100 c c serum mixed with equal volume of sterile saline mixture about blood temperature Repeat hourly until temperature reaches 102°

PROPHYLACTIC INOCULATION Pneumonia is prevalent among natives employed in South African mines The incidence and mortality are highest during the first weeks of employment Lister finds that prevalent strains agree with Types I, II and IV of the Rockefeller Institute and has prepared a vaccine dosage three weekly injections with total of 000 million cocci Considerable immunity results

MORBID ANATOMY

The changes of acute inflammation occur in the lung but so modified by the nature of the tissue as to be characteristic three stages are recognized (1) Engorgement (2) Red hepatization (3) Gray hepatization and also (4) Resolution

(1) Stage of Engorgement.

MACROSCOPIC Lung deep red firm, and more solid than normal On section surface red and moist Air present and lung crepitates but less than normal Portions float in water

HISTOLOGY Capillaries dilated and engorged Alveoli contain some blood corpuscles, alveolar cells, and serum Alveolar epithelium swollen

(2) Stage of Red Hepatization. —

MACROSCOPIC Lung appears bulky and feels heavy Is firm and airless Pleurisy present on surface On section surface is red brown, dry, and granular (due contents of alveoli) Distinctly friable Does not crepitate Sinks in water On scraping surface, small amount of reddish exudate (containing numerous diplococci)

HISTOLOGY —Alveolar spaces occupied by network of coagulated fibrin containing red and white blood cells and occasional epithelial cells Alveolar walls infiltrated, and some leucocytes present in interlobular tissues.

Lobar Pneumonia—Morbid Anatomy, *continued.*

3. Stage of Gray Hepatization.—

MACROSCOPIC.—*Colour gray.* On section surface moister and granules indistinct. Extremely friable. Does not crepitate. Sinks in water.

HISTOLOGY.—Alveolar spaces filled with leucocytes (in preparations, the plug is often retracted from the wall). Fibrin and red cells have been removed by phagocytic action of leucocytes.

In extreme cases, this stage is sometimes called 'purulent infiltration'. Surface of cut lung covered with a purulent fluid.

4. Resolution.—Proteolytic enzymes digest and liquefy the alveolar contents, and the product is mainly absorbed and excreted by the kidneys. Some leucocytes are ejected in the sputum.

Distribution of Lesions in the Lungs.—(1) One lung alone is commoner than both. (2) Right lung is commoner than left. (3) Base is commoner than apex; commences at base in 75 per cent. (4) When both lungs affected, is usually both bases; both apices is rarest combination; the middle lobe is very rarely affected alone. (5) Several lobes may be affected simultaneously, or, more frequently, in succession, various stages being present at same time. (6) Apical pneumonia is commoner in children than adults; under five years, apices only in 30 per cent. (7) Central pneumonia, commencing at root, is rare (and doubtful).

STATISTICS: (1) Right lung only, 55 per cent; left only, 25 per cent; both, 20 per cent. (2) One lobe, 40 per cent, two lobes, 40 per cent; more than two, 20 per cent.

WEIGHT of consolidated lung about 50 ounces (normal about 20 ounces).

AREA OF LUNG NOT CONSOLIDATED. Rarely normal. Usually congested and œdematous. The unaffected lung is usually congested: compensatory emphysema common.

PLEURA.—Inflammatory changes invariably present where pneumonic process has reached surface.

BRONCHI.—Contain froth; rarely the thick mucus of pneumonic sputum.

BRONCHIAL GLANDS.—Swollen; suppuration extremely rare.

Lesions in Other Organs.—Not common.

HEART.—Often contains firm coagula, especially on right side.

PERICARDITIS.—Commonest cardiac lesion (*see* COMPLICATIONS).

ENDOCARDITIS.—Rare, usually ulcerative. Pneumonia is a frequent antecedent in deaths from ulcerative endocarditis.

Less important changes are slight enlargement of the spleen and changes in the kidneys.

Rare occurrences are meningitis and colitis.

SYMPTOMS.

Incubation Period.—Unknown. Probably few hours to few days.

General Description.—

ONSET.—Abrupt, with rigor. *Temperature* has already risen during chill. General sensations of a severe febrile attack.

PRESENT FROM ONSET OR DEVELOPING RAPIDLY.—(1) Pain in the side, often very severe; (2) Short dry cough; (3) Rapid respiration.

DISEASE FULLY DEVELOPED.—Within twenty-four to forty-eight hours, condition characteristic: –

1. FACE flushed and eyes bright. Expression anxious.
2. RESPIRATION.—Short and rapid, frequently an *expiratory* grunt, or pause after expiration. Alæ nasi dilate.
3. COUGH. –Short, frequent, and repressed. Increases pain in side
4. EXPECTORATION. –Very tenacious and blood-stained ('rusty sputum').
5. SKIN. Dry and pungent.
6. PULSE. Full and bounding Pulse respiration ratio often [illegible]
7. LABIAL HERPES. - common
8. TEMPERATURE.—High 104 common.
9. PHYSICAL SIGNS IN LUNGS.

TERMINATION. –In typical cases by crisis, after five to ten days. Rapid convalescence.

Special Features. -

1. VARIETIES OF ONSET. May be less abrupt than usual: patient may remain at work until lung is solid. Onset tends to be more insidious in elderly or debilitated persons, and in terminal pneumonia.

More than one rigor is rare only in severe attacks

2. THE FEVER.—

a PERIOD OF RISING TEMPERATURE *Initial rise* very rapid: frequently reaches 102 to 104° F. in a few hours. Rise above 104° at onset not necessarily serious. Probably is evidence of healthy reaction *Variations in rise of temperature* occur in children; in absence of a chill, rise is often more gradual; in drunkards, and in weakly and old people, temperature does not rise so high or so rapidly prognosis bad; also when pneumonia occurs as a complication in other diseases.

b. PERIOD OF CONTINUED TEMPERATURE (FASTIGIUM).—*Temperature usually very constant*: variations often do not exceed 2°. Continuous high temperature, over 104°, is severe but not necessarily serious; in fatal cases may rise further or fall suddenly before death. Lower temperatures may be mild cases or due to poor reaction of system. Slow gradual fall from high temperature at onset is often serious.

c. PERIOD OF FALLING TEMPERATURE. The temperature falls either *by crisis or by lysis.* If defervescence occupies longer than thirty-six hours, it is considered as lysis.

Crisis.—Temperature falls abruptly. Occurs most commonly between 5th and 10th days, especially on 7th. Rare after 12th, and not before 3rd day. Complete before 9th day in 90 per cent. *Fall* occupies 6 to 12 hours: 24 hours is a protracted crisis. Crisis probably

Lobar Pneumonia - Symptoms—Special Features, *continued.*

marks stage of active immunity to toxins of pneumococcus: no evidence of occurrence of phagocytosis. *Profuse sweating* frequently precedes fall of temperature; patient then falls asleep; on waking, temperature, dyspnœa, general symptoms, and distress have abated without corresponding changes in physical signs. *Temperature curve at time of crisis* may show one or more of the following stages: (i) Pseudo-crisis: temperature falls nearly to normal and rises again: crisis follows, usually in 24 to 48 hours. (ii) Pre-critical rise: rises slightly shortly before crisis. (iii) Crisis: often falls to subnormal. (iv) Post-critical rise: rises slightly next day.

Lysis.—Temperature falls more gradually. More common in children (in 30 per cent of cases) than in adults. Usual form after 12th day of fever. In cases of delayed resolution, fever may persist many weeks.

3. PAIN.—Early symptom, rarely absent; often extremely severe; worse on coughing and deep inspiration.

CAUSE.—Due to involvement of pleura—therefore absent in central pneumonia, slight in apical, and most severe when the diaphragmatic pleura is affected.

NATURE OF THE PAIN.—

(a) *Local Pain.*—Almost invariable. Over area of affected pleura: deep tenderness on pressure: no superficial tenderness.

(b) *Referred Pain.*—Not uncommon. If the inflammation affects the intercostal trunks, pain is referred to their terminal distribution; hence pain is felt *in abdomen or iliac fossa.* Superficial tenderness absent.

(c) *Reflected pain* from the lung. Pathological changes in lung render nerve end-organs incapable of stimulation; hence reflected visceral pain is extremely rare. When it occurs, pain and superficial tenderness are present and may be on opposite side to affected lung.

4. DYSPNŒA.—Practically constant, from onset.

RATE OF RESPIRATION.—In adults: usually 40 to 50 when condition developed: at onset about 30. In children: 55 to 60—over 70 bad prognosis.

CHARACTER OF RESPIRATION.—Shallow and restrained. *Expiratory grunt* frequent. Inverted rhythm not uncommon in young children.

COURSE.—Marked increase during febrile period usually means bad prognosis. *At crisis,* rate falls, but more slowly than pulse and temperature: often several days before reaching normal.

CAUSE.—Many factors are present: (a) *Toxæmia* is the main cause; (b) *Pain* causes shallow, and therefore rapid, respirations, of jerky character; (c) *Fever* is of little importance; (d) *Consolidation of lung* is of some importance, but degree of dyspnœa is largely independent of amount of consolidation.

PULSE-RESPIRATION RATIO (normally 4 : 1). Lower than in any other condition; often 2 : 1; in children may be 1 : 1.

CYANOSIS.—Slight degree common: in toxæmia lividity marked. Extreme cyanosis may develop in severe conditions, but, in general, cyanosis is less prominent than in bronchopneumonia.

5. COUGH.—An early symptom—onset usually with the pain. Typically: short, restrained, and frequent. Pain and distress often extreme. Disappearance of cough with signs of secretion in bronchi is a serious symptom. Often absent in old and young people and drunkards, and in terminal pneumonia. *After crisis*, becomes looser and less distressing.

6. SPUTUM.—At onset may be clear and mucoid: *very tenacious and of small amount* throughout.

'RUSTY SPUTUM.' Usually present within two days. Occurs in more than half the cases. *Extremely tenacious.* No air-bubbles. Does not mix with saliva or pus. *Amount small*, one to two ounces a day. Colour due to blood, and gradually disappears. After crisis, sputum becomes looser, and often more profuse.

IN CHILDREN (occasionally up to eleven years of age), often no expectoration owing to swallowing of sputum; occasionally rusty sputum is vomited. In old people also there may be no sputum.

HÆMOPTYSIS.—Occasionally brisk at onset: usually several ounces. Is not of bad prognosis, and not necessarily due to tuberculosis or cardiac disease.

COMPLICATIONS, e.g., bronchitis or œdema of lung, may alter character of sputum.

MICROSCOPICAL CHARACTERS.—Leucocytes, red cells, mucus, epithelial cells, and various micro-organisms. May be fibrinous plugs of smallest bronchioles. Chemically: rich in *calcium chloride*.

7. POSTURE.—Varies. Patient usually lies on affected side.

PHYSICAL SIGNS IN THE LUNGS.

INSPECTION.—Movements of affected part are deficient; site often obvious when lesion extensive. When lower lobe affected, apex may move more freely than normal. Movement of healthy lung increased. Visible cardiac pulsation may be increased when left upper lobe is affected.

Note also rate of respiration, and action of accessory muscles of respiration.

PALPATION.—Lack of expansion of affected site. *Vocal fremitus* increased unless bronchi filled with secretion. (Patient should cough before test.)

Percussion and auscultation vary greatly in the different stages:—

Stage of Engorgement.—

PERCUSSION.—Little change, or note high-pitched and tympanitic and by comparison may appear dull.

Lobar Pneumonia—Physical Signs in the Lungs, *continued.*

AUSCULTATION.—(1) Breath-sounds weak (often the earliest physical sign); (2) Fine 'crepitant râles'.

The 'crepitant râles' appear close to ear, occur towards end of respiration, often only on deep breathing, but not removed by coughing: probably due to separation of walls of alveoli stuck together lightly by exudation, but possibly are of pleural origin. The breath-sounds, rarely, are harsher on affected side.

Stage of Hepatization (Consolidation).—

PERCUSSION.—Note dull. Quality and degree vary considerably. Resistance to finger and woody dullness of fluid not present.

AUSCULTATION.—(1) Tubular breathing. Bronchial breathing commences with low pitch during expiration: as consolidation develops, rapidly increases to characteristic 'tubular breathing', *intense high-pitched, continuous* throughout inspiration and expiration, *with complete absence of adventitious sounds.* (2) Bronchophony, viz., vocal resonance greatly increased. No adventitious sounds present during height of stage.

Stage of Resolution.—Physical signs commence to change usually within twenty-four hours of crisis.

PERCUSSION.—Note gradually returns to normal.

AUSCULTATION.—Tubular breathing gradually disappears. May be 'redux crepitations', but often absent. Lungs return to normal in four to seven days: in children sooner. When consolidation has been extensive, percussion note for several weeks may remain abnormal, slightly dull or tympanitic. When temperature falls by lysis, resolution is usually slower. Occasionally consolidation appears to spread after crisis. Possibly crisis marks a general immunity but a local immunity does not occur simultaneously.

Physical Signs in Unaffected Lobes or Lung.—(1) Movement increased; (2) Percussion note hyper-resonant; (3) Breath-sounds loud and puerile in character (4) No moist sounds unless bronchitis or congestion is present. (The lesion is occasionally diagnosed on the wrong side in the early stages.)

Central Pneumonia.—Occasionally symptoms are typical, but physical signs are absent or develop later. Explanation may be: (i) Central pneumonia commencing near root. *Never found post mortem*, but is suggested by radiographs. (ii) Symptoms due to pneumococcal septicæmia, and lungs involved later: probable explanation.

CHANGES IN OTHER SYSTEMS.

Circulatory System.—

PULSE.—*Full and bounding.* Rate increased in proportion to pyrexia, 100 to 120. Not dicrotic. *Variations of pulse without cardiac failure*; children faster than adults, 120 to 160; healthy young adults often under 100; in old and feeble persons, small and rapid from onset; with extensive consolidation, may be

small and running. Even in serious cases pulse may be full and deceptive in prognosis. After crisis, rapidly becomes normal. Bradycardia occasionally during convalescence; is of no significance.

HEART SOUNDS.—Usual variations from normal are: (1) Sounds loud and clear; (2) Pulmonary second sound accentuated; (3) Mitral and pulmonary murmurs not uncommon during fever, especially in children.

FAILURE OF THE HEART.—The possibility is a constant anxiety. *Early physical signs* of failure: disappearance of accentuated pulmonary second sound; dilatation of right side of heart; sounds develop fœtal rhythm. Pulse-rate usually increases. *Symptoms*: Increasing cyanosis, orthopnœa, and diminution of urine. *Collapse with rapid feeble pulse* may occur early not always fatal. Rapid cardiac failure with toxæmic symptoms may occur suddenly and fatally, in healthy people: very rare.

ENDOCARDITIS AND PERICARDITIS.—(*See* COMPLICATIONS.)

BLOOD-PRESSURE.—No constant variation: often unchanged throughout attack and crisis. Gradual fall of more than 20 mm. Hg suggests cardiac failure. Prognosis serious if pulse-rate per minute exceeds blood-pressure in mm. of Hg.

BLOOD.—*Leucocytosis* appears early: number 12,000 to 25,000 per c.mm., rarely exceeds 30,000 per c.mm. Polynuclear cells: increase in percentage. Returns gradually to normal after crisis. *Prognosis* is most favourable with moderate leucocytosis (about 15,000 per c.mm). Serious in absence of leucocytosis. Anæmia is unusual.

Skin.—*Hot and pungent.* Important changes are: (1) *Herpes* more common than in any other fever: in 25 per cent of cases. Site: around mouth and nose; very rarely elsewhere. Prognosis favourable when present. Cause unknown. Pneumococci said to have been isolated from vesicles (2) *Sweats*: profuse at crisis, often slightly precede fall of temperature. Not common during fever. Subsequent to crisis suggest suppuration (empyema).

Digestive System.—No change distinctive from other fevers.

TONGUE.—Commonly white and furred. Dry in toxæmia.

APPETITE.—Lost early. Recovers rapidly after crisis.

VOMITING.—Rare except in children.

BOWELS.—Usually constipated: may act normally. Diarrhœa rare. Meteorism: occasionally severe.

SPLEEN.—Not uncommonly enlarged (examination difficult owing to pain on deep respiration).

Urine.—Usual febrile characters present. *Trace of albumin* common. Albumose in severe cases. *Excretion of chlorides markedly diminished*: true retention occurs, retained sodium chloride being excreted after crisis (Hutchison); no apparent value in prognosis. Acute nephritis rare.

Nervous System.—Most frequent symptoms are:—

1. HEADACHE.—Occurs in 50 per cent. Rarely severe.

Lobar Pneumonia—**Nervous System,** *continued.*

2. INSOMNIA.—Frequent, *often severe and extremely troublesome to treat.* Aggravated by, but may be entirely independent of, pain, cough, or dyspnœa.
3. DELIRIUM AND PSYCHICAL DISTURBANCES.—Slight degrees of mental dullness rarely absent in typical forms. With severe delirium and psychical disturbances, prognosis is serious. Occurs in: (*a*) Toxic cases. (*b*) Delirium tremens in alcoholic patients. Very common. (*c*) Onset with acute mania (rare). (*d*) Onset in children simulating meningitis; prognosis is not serious.

   Apical pneumonia is more liable to nervous symptoms. Cerebral symptoms occasionally occur after crisis. Recovery in all forms is rapidly complete when not fatal.
4. CONVULSIONS IN CHILDREN. May occur: (*a*) At onset in place of rigor; (*b*) Repeatedly at onset in cases simulating meningitis; (*c*) Later in attack at commencement of true meningitis (rare).

COMPLICATIONS.

Complications are few in number, but account for a considerable percentage of fatalities. The most important are: (1) *Pleurisy and empyema*; (2) *Pericarditis*; (3) *Endocarditis*; (4) *Meningitis.*

Bronchitis in some degree is almost constant, and is part of the disease.

1. **Pleurisy and Empyema.**—

PLEURISY is practically a part of the disease: inevitable when inflammation reaches surface of lung. Thickened pleura, sufficient to give signs, very rarely follows pneumonia (and is not usual explanation of persistent impaired resonance).

EMPYEMA is most common complication; about 4 per cent of cases. Commoner in children: about 12 per cent, and in 30 per cent of fatal cases.

BACTERIOLOGY.—Pneumococcus commonest, and best prognosis. Streptococcus not infrequent, especially in adults. Staphylococcus and other organisms very rare.

ONSET AND SYMPTOMS.—(i) Temperature rises again one to four days after subsidence; (ii) Sweats; (iii) General malaise, cough may return; (iv) Leucocytosis. Pain, dyspnœa, and rigors are unusual. Temperature may not fall to normal, but commences to rise again during lysis.

PHYSICAL SIGNS.—Those of pleural effusion. Vary with amount of fluid, which may be small.

Interlobar or diaphragmatic empyema is *very rare.*

2. **Pericarditis.**—In about 1 per cent: in more than 10 per cent of fatal cases (statistics vary greatly). Mortality at least 80 per cent in diagnosed cases. Often insidious and undiagnosed, hence frequency in recoveries cannot be estimated. *Amount of fluid* rarely exceeds a few ounces. More common with right than left pneumonia; origin probably septicæmic, but may be direct

extension. Pleurisy almost always present. Occurs usually in types otherwise severe. Physical signs of pericarditis, but obscured by pleural friction and pulmonary signs.

3. **Endocarditis.**—Rare. Is practically always ulcerative. Commoner in women. Specially affects hearts with previous valvular trouble. Aortic valve commonest, but right side more often affected than in other forms of endocarditis.

MENINGITIS is common termination.

4. **Meningitis.**—Rare, but always fatal. Occurs in about 2 per cent of children under 10 years. *In adults very rare.* Onset at height of fever, affects the vertex, and is usually not diagnosed, symptoms being ascribed to toxæmia. Occasionally later in disease affects base and may be diagnosed. Pneumococci present in cerebrospinal fluid. May occur later with endocarditis following pneumonia.

Other Complications.—

1. PULMONARY COMPLICATIONS include abscess and gangrene of lung, considered under MODES OF TERMINATION (p. 61).
2. COMPLICATIONS RESULTING FROM PNEUMOCOCCAL SEPTICÆMIA.—Commoner in children. Onset usually a few days after temperature becomes normal or during fall by lysis. Meningitis certainly, and probably pericarditis and endocarditis, described above, really belong to this group.
   - *a.* OTITIS MEDIA.—Not uncommon in children: in 3 per cent of cases. No special characteristics.
   - *b.* ARTHRITIS. Mainly in children. May precede onset of pneumonia. Larger joints affected; hot painful swelling; in mild cases may subside, in severe cases suppuration may occur. Mortality very high in latter probably from associated septicæmia.
   - *c.* JAUNDICE.—Slight icteroid tinge not uncommon, but definite jaundice rare. Cause doubtful. Usually slight, begins during pyrexia, and prognosis good. In toxæmic cases, mortality high.
   - *d.* PERITONITIS.—Very rare. Onset follows defervescence. Mortality very high. (*See* PERITONITIS.)
   - *e.* NEPHRITIS.—Rare.
3. VARIOUS AND RARE COMPLICATIONS.—

   THROMBOSIS.—Occurs rarely in peripheral veins, usually femoral. Ante-mortem clots in the heart are very rare.

   EPISTAXIS.—May occur at onset (about 3 per cent).

   COLITIS.—In severe cases.

   Appendicitis may co-exist, but relationship is doubtful.

   Peripheral neuritis, aphasia, parotitis, and numerous other complications occasionally recorded.

RELAPSES AND RECURRENCES. CONVALESCENCE.

Relapse.—Different lobes may be successively involved (creeping pneumonia). True relapse after crisis is extremely rare; initial attack usually abortive.

Lobar Pneumonia, *continued.*

Recurrence.—Very common in pneumonia—immunity due to attack is of short duration.

Convalescence.—Generally rapid and uninterrupted. Sequelæ rare.

CLINICAL VARIETIES.

Anatomical Varieties.—(*See* DISTRIBUTION IN LUNGS, p. 52.)

APICAL PNEUMONIA.—Commoner in children: said to be frequently associated with cerebral symptoms.

CREEPING PNEUMONIA.—Involving successive lobes.

DOUBLE PNEUMONIA.—Affecting both lungs simultaneously, usually bases. In latter, case mortality high.

CENTRAL PNEUMONIA.

MASSIVE PNEUMONIA.—The bronchi as well as alveoli are filled with exudate. Extremely rare. Physical signs resemble effusion.

Varieties Associated with Age.—

PNEUMONIA IN CHILDREN.—Main variations from adult type are: *Rigor rare*, onset frequently with convulsion. *Sputum absent*, is swallowed. *Apex* not uncommonly affected: 30 per cent of cases. Cerebral symptoms frequent. *Empyema* commoner than in adults. *Septicæmic complications* commoner. General mortality very low: about age of 3 years, death is very rare.

PNEUMONIA IN THE AGED. Onset, symptoms, and physical signs all indefinite. Prostration marked and mortality high.

Other Varieties.—The following varieties may also be considered:

ALCOHOLIC SUBJECTS.—Pneumonia extremely common in drunkards. Condition resembles delirium tremens. Onset, symptoms, and physical signs of pneumonia often indefinite. Mortality high.

TERMINAL PNEUMONIA.—Pneumonia may be the terminal condition in chronic diseases such as diabetes, heart disease, nephritis, or phthisis. Symptoms and physical signs are slight.

SECONDARY OR INTERCURRENT PNEUMONIA.—Not uncommon in certain specific fevers, e.g., typhoid fever. Symptoms indefinite. Physical signs slight: percussion note impaired, breath-sounds feeble, few crepitations. Bases usually affected. Histologically may be lobular pneumonia.

EPIDEMIC PNEUMONIA.—Definite epidemics occur: generally marked by special features and high mortality. Organisms other than pneumococcus may be the cause, e.g., plague bacillus. Certain of these epidemics are related to influenza.

LARVAL OR ABORTIVE PNEUMONIA.—Mild cases or with very short duration.

ASTHENIC, TOXIC, OR 'TYPHOID' PNEUMONIA.—Local lesions slight. Prominent symptoms are suggestive of septicæmia, viz., prostration, marked nervous and toxæmic condition, jaundice, gastro-intestinal symptoms. Probably is pneumococcal septicæmia: pneumococci often isolated from blood. Must not be confused with pneumonia occurring as a complication of typhoid fever.

POST-OPERATIVE PNEUMONIA.—Is now rare. Frequency much reduced by: (1) Use of ether by open method; (2) Improved surgical technique; (3) More rapid preparation of skin by iodine, etc. Symptoms indefinite. Physical signs of low pneumonia: impaired resonance, feeble breath-sounds, crepitations. Post-operative consolidation of the lungs is not always a true lobar pneumonia, but may be divided into:—

1. INHALATION OR ANÆSTHESIA PNEUMONIA.—Probably caused by aspiration of saliva, etc. Cooling of lungs by vapour of less importance.
2. HYPOSTATIC PNEUMONIA.—Influenced by recumbent posture, feeble circulation, and interference with diaphragm. Commoner after abdominal operations.
3. MASSIVE COLLAPSE OF THE LUNGS.
4. PULMONARY EMBOLUS.

Association of Pneumonia with other Diseases.—

1. TUBERCULOSIS.—Phthisis often terminates with a lobar pneumonia. Onset of acute tuberculous pneumonia may simulate lobar pneumonia. Lobar pneumonia never terminates in tuberculosis. cases where this appears to occur have been tuberculous from onset. There is no evidence that lobar pneumonia predisposes to tuberculosis.
2. INFLUENZA.—*See* INFLUENZA.
3. TYPHOID FEVER.—Pneumonia may occur at onset or in third week of typhoid fever. (*See* p. 15.)
4. INFECTIOUS DISEASES. Scarlet fever: pneumonia rare, but mortality high. Measles, whooping-cough, typhoid, etc.
5. EMPHYSEMA AND CHRONIC BRONCHITIS.—Increase severity of attack and prognosis of lobar pneumonia: death occurs in two or more weeks.
6. MALARIA.—May co-exist with pneumonia, and symptoms of both become confused. Otherwise diseases are independent.

MODES OF TERMINATION.

Pneumonia may terminate as follows: (1) *Resolution;* (2) *Delayed resolution;* (3) *Organization and fibrosis—chronic interstitial pneumonia;* (4) *Abscess;* (5) *Gangrene.*

1. RESOLUTION.—Of cases which recover, 90 per cent terminate by normal resolution: 60 per cent after crisis, and 30 per cent after lysis. Lung usually normal within two weeks, frequently seven to ten days. The exudate in the alveoli mainly removed by liquefaction and absorption by the blood: some plugs may be coughed away. Resolution occasionally, but rarely, occurs without sputum.
2. DELAYED RESOLUTION.—About 4 per cent of all cases. Lower lobe usually involved, especially right. *Duration:* Rarely exceeds six weeks.

   CLINICAL COURSE.—Crisis or lysis occurs, but temperature usually does not entirely subside. Physical signs of consolidation persist, usually over small area. After

Lobar Pneumonia—**Modes of Termination,** *continued.*

varying period, resolution occurs, generally slowly. Organization may follow. Pleural effusion must be excluded, and sputum examined for tuberculosis. Condition is not confined to debilitated persons, but in these and drunkards may be fatal.

Cause: probably failure of autolytic action of body fluids. Leucocytosis is absent.

3. CHRONIC INTERSTITIAL PNEUMONIA. Very rare. The exudate organizes, resulting in fibrosis of the lung or chronic interstitial pneumonia (q.v.). Delayed resolution precedes the fibrosis.
4. ABSCESS. Rare termination. Mortality high. Onset insidious. *Symptoms severe*: intermittent or remittent pyrexia, cough often severe and paroxysmal, sputum contains pus and elastic tissue, and becomes offensive. Signs of consolidation or excavation. (*See* ABSCESS OF LUNG.)
5. GANGRENE.—Extremely rare. Practically always fatal. Often with abscess. Sputum unbearably fœtid: usually renders diagnosis certain. Abscess and gangrene occur most frequently with *diabetes.*

DIAGNOSIS.

Usually simple. In the first stage, symptoms may be practically conclusive before physical signs admit of localization. Difficulties in diagnosis arise from: (1) *Conditions in which the onset and nature of the attack are modified*; (2) *Conditions in which confusion with other diseases occurs.*

1. **Onset and Nature of Attack Modified.**—In terminal, secondary, intercurrent pneumonia in other diseases, and pneumonia in the aged. The condition is a 'low' pneumonia, viz., indefinite onset with physical signs not very marked. Condition is more frequently overlooked in these circumstances than an erroneous diagnosis made. Its onset is suggested by *rising temperature and cough*, and signs in lungs on examination.

IN CHILDREN.—Difficulty especially arises from:—

a. CEREBRAL SYMPTOMS IN PNEUMONIA.

b. PLEURISY WITH EFFUSION SIMULATING PNEUMONIA. In children, vocal fremitus and tubular breathing may be present. Diagnosis mainly by hypodermic needle.

c. VARIOUS EXANTHEMATA.—Convulsion and general condition at onset may be similar.

d. CONFLUENT BRONCHOPNEUMONIA.

e. APPENDICITIS AND ACUTE ABDOMINAL CONDITIONS.

IN ALCOHOLIC SUBJECTS.—Obscured by delirium tremens.

2. **Confusion with other Diseases.**—This occurs in:—

a. ACUTE ABDOMINAL DISEASES.—When the pleuritic pain in pneumonia is referred to the abdomen, the abdominal wall becomes rigid and tender. Condition at onset may simulate almost any acute abdominal lesion. Diagnosis important, as operation may be suggested. Difficulties most often with:—

APPENDICITIS —Diagnosis limitation of movement of chest, pulse respiration ratio, and early signs of pneumonia This difficulty is especially frequent in children, owing to vomiting in pneumonia

PERFORATED GASTRIC ULCER

b ACUTE PNEUMONIC PHTHISIS Diagnosis at onset often impossible Defervescence does not occur or is not complete pyrexia becomes irregular, wasting, consolidation persists. Later, tubercle bacilli in sputum Fatal in two weeks or upwards Suspect when temperature persists after twelfth day, but remember is *very rare*

c TYPHOID FEVER —Difficulties may arise from (*a*) Typhoid state developing in toxic pneumonia (*b*) Pneumonia occurring as complication in typhoid fever at onset or in third week Diagnosis often depends on distinctive proofs of typhoid fever, i e, rash and agglutination reaction (The spleen is frequently enlarged in pneumonia)

d INFLUENZA

e. ABNORMAL FORMS OF LOBAR PNEUMONIA —If several cases occur in same household and *all are rapidly fatal*, possibility of plague must be considered

Note —Pneumococcus may often be isolated from blood cultures early in pneumonia

PROGNOSIS.

General Mortality. The mortality of all cases at all ages is from 20 to 25 per cent In private practice considerably less than in hospitals The prognosis varies greatly in different circumstances depending mainly upon (1) *Age*, (2) *Previous habits and conditions of health*, (3) *Features of the attack*

✓ 1 AGE Under 2 years mortality is high but the disease is rare Between 2 and 5 mortality is extremely low, an uncomplicated pneumonia rarely dying, recovery not uncommonly occurs even when child appears moribund Mortality increases progressively with age between 20 and 30 years about 20 per cent at 60 years about 60 per cent

✓ 2 PREVIOUS HABITS AND CONDITIONS OF HEALTH — There is no disease where previous conditions of life are so important in prognosis In healthy young adults fatalities are rare The most important factors are —

a ALCOHOL More than doubles mortality Especially seen in middle aged labourers

b DEBILITATING DISEASES —Especially chronic nephritis diabetes, cardiac disease, arteriosclerosis, and phthisis

c POOR PHYSIQUE, previous insufficient food and unhealthy surroundings Death rate is higher in cities than in country districts. *Stout individuals* are bad subjects.

✓ 3 FEATURES OF THE ATTACK Conditions influencing prognosis may be considered under the headings General symptoms and signs, extent of pulmonary lesion; varieties of termination, complications.

Lobar Pneumonia—**Prognosis**, *continued.*

GENERAL SYMPTOMS AND SIGNS (approximately in order of importance).—

a. Toxæmia.—The degree of toxæmia is the most important sign in an attack.

b. Condition of the heart and pulse.—Especially dilatation of right side of heart and rapid small pulse. *If pulse exceeds 130, prognosis is serious*; also if pulse-rate per minute exceeds blood-pressure in mm. of Hg.

c. Delirium.—When marked.

d. Pyrexia.—Duration of considerable importance; degree less so in general. Extremes are serious, viz., hyperpyrexia, over 106°, and low temperature with toxæmia (poor reaction).

e. Dyspnœa.—Rate over 50 is serious, or *pulse-respiration ratio* falling to 2 to 1.

f. Insomnia.—When intractable.

g. Leucocytosis.—Absence is bad sign.

Usually two or more of above co-exist, e.g., with toxæmia, commonly rapid pulse, extremes of temperature, absence of leucocytosis. These conditions may be extreme when extent of consolidation is very slight.

EXTENT OF PULMONARY LESION.—Mortality increases with the number of lobes affected: indicating severity of intoxication. *Death is rarely due to asphyxia* from extent of lung involved; toxæmia and cardiac failure are the usual direct causes.

VARIETIES OF TERMINATION.—Abscess and gangrene have very high mortalities. With delayed resolution, exhaustion and cardiac failure may occur.

COMPLICATIONS.—Present in high percentage of fatal cases.

Empyema: the least serious, but, as with all complications, *the earlier the onset in the attack, the worse is the prognosis.* All other complications have high mortality.

Meningitis: Always fatal.

Endocarditis and pericarditis: High percentage.

PNEUMONIA AND PREGNANCY.—Mortality is higher in pregnancy, especially in later months. Abortion is common (at least half), and increases in later months, and raises the mortality; and the earlier in the attack it occurs, the higher the mortality. The liability to pneumonia is not increased by pregnancy.

TREATMENT.

There is no specific drug, and the pulmonary inflammation probably is not influenced by any treatment. The aim of treatment is, in general, to maintain the strength, and, in particular, to deal with special symptoms.

General Principles of Treatment.—

1. GENERAL MANAGEMENT.—Free ventilation in room. Confined strictly to bed. Clothing warm but not heavy: a Gamgee

or woollen jacket is very suitable, but not essential. Hot-water bottle to feet. Daily sponge with tepid water. In suitable climates should be in the open air. The mouth must be carefully cleansed.

2. DIET.—*Water*, lemonade, or bland fluids must be given freely. In delirium, saline infusions per rectum, or intravenous injections. *Food*: milk, 3 pints in 24 hours at intervals of 2 to 3 hours. Milk sugar, eggs, Mellin's or cereal foods may be added.
3. BOWELS.—At onset give calomel and follow with a saline aperient. Give salines or enemata during febrile period. *Purging is inadvisable*: may start diarrhœa. Meteorism treated by turpentine enema, or turpentine stupe or pituitrin.
4. BLEEDING.—Of greater value in pneumonia than in any other condition. In full-blooded patients, should be routine at onset; later, is indicated by dilatation of right heart or cyanosis. Most efficacious from the jugular vein. Amount: 15 to 20 ounces.
5. HYDROTHERAPY.—Is best treatment for toxæmia and cardiac failure; cold sponging every three hours. Disturbance necessary for full baths is rarely advisable.
6. ANTISERUM AND SPECIFIC THERAPY.—(*See* BACTERIOLOGY.) Vaccine treatment: at present, no proof of value exists.

Symptomatic Treatment.—

1. RELIEF OF PAIN.—Hot poultices to the side, or an ice-bag. Leeches. For morphia, *see below*.
2. TOXÆMIA.—*Alcohol* (brandy or whisky) 4 to 8 ounces daily: also cardiac stimulants. *Hydrotherapy. Water free* by mouth. *Intravenous saline injections* or continuous saline by rectum. *Bowels* freely opened by saline aperients.
3. CARDIAC WEAKNESS.—*Alcohol and hydrotherapy. Cardiac stimulants*: (*a*) Camphor, hypodermically (camphor gr. ij dissolved in ♏x of sterile olive oil, eight-hourly). (*b*) Caffeine hypodermically (caffeine sodium salicylate gr. iij, eight-hourly) may be given alternately with camphor. (*c*) Injections of strychnine. (*d*) Digitalis by the mouth. Drugs should not be given as a routine to support the circulation. Strychnine, alcohol, and digitalis may be given earliest: camphor and caffeine are most rapid, and of great value for urgent treatment
4. TO ASSIST THE RESPIRATORY SYSTEM.—The patient can usually choose the most comfortable position. Avoid all exertion and relieve pain. Free ventilation. Expectorant drugs are of little value for the cough, and preferably omitted. For severe cough, heroin may be given (for linctus, *see* BRONCHITIS). *Venesection* if right heart is dilated.
5. INSOMNIA.—Often extreme, and difficult to treat, while sleep is essential. Paraldehyde ʒj hourly up to ℥j is frequently effective, or ʒij repeated after one hour (prescribe in ℥ss to ℥j whisky and ℥ij water). Chloral hydrate and trional are more depressant.

Lobar Pneumonia—Treatment, *continued.*

6. MORPHIA.—The question of administration is very important. The following general statements may be made. (1) The pain, or, far more important, the insomnia, often yields to no other drug; (2) Sleep is often essential; (3) It is certainly dangerous to give morphia at the crisis; (4) It is impossible to foretell when the crisis will occur. On these it may be observed: *First*—It is safe to give morphia at the onset. Injection of morphia, gr. ¼, is good treatment up to five days from onset. *Secondly*—It is dangerous to repeat a hypodermic injection of morphia. *Finally*—Every case must be considered separately, but in doubtful cases it may be remembered that post-mortem findings suggest that most cases which die after morphia would have had a fatal ending in any event.
7. DELIRIUM.—Alcoholic subjects. *Careful watching*, ice-bags to the head, cold packs, and cold sponges. With patients who have been heavy drinkers, alcohol should be commenced at once, conveniently given as stout.
8. CRISIS.—Collapse, cardiac and respiratory, must be watched for. Atropine gr. $\frac{1}{100}$ hypodermically, with strychnine gr. $\frac{1}{60}$, for profuse sweating in a feeble patient.
9. HYPERPYREXIA.—*Hydrotherapy.* ANTIPYRETICS ARE CONTRAINDICATED.
10. DELAYED RESOLUTION.—When apyrexial, give respiratory exercises, or blow-bottles. Rest during pyrexia.
11. OXYGEN.—The value of oxygen in the past has been disadvantageously affected by late employment and inefficient methods. May prove to be of great value when administered in measured dosage over long periods, as by *Haldane's apparatus* (advantage of great simplicity in use): in absence of this, with Leonard Hill's mask (strong dilutions for short periods).

Convalescence.—Is extremely rapid. In normal cases, patient may be allowed up in a week and regarded as cured in a fortnight. If cardiac failure has occurred, convalescence must be more gradual.

BRONCHOPNEUMONIA.

(*Capillary Bronchitis. Catarrhal Pneumonia. Lobular Pneumonia.*)

A bacterial infection commencing with inflammation of the bronchioles and extending to the alveoli. Groups of alveoli become filled with cells, mainly by desquamation from the walls.

ETIOLOGY.

Occurs as a primary or a secondary condition. In a third group are cases of aspiration or deglutition pneumonia.

Primary Bronchopneumonia.—Closely resembles lobar pneumonia in etiology, and also in symptoms. Majority of cases in children under 2 years, rare over 4 years.

Secondary Bronchopneumonia.—The following conditions may precede or be predisposing causes:—

1. BRONCHITIS.—The inflammatory process spreads down from the bronchi to the bronchioles.

2. ACUTE SPECIFIC FEVERS.—Especially measles, whooping-cough, and influenza; less commonly diphtheria, scarlet fever, and typhoid fever.
3. RICKETS AND DIARRHŒA IN INFANTS.
These three groups are extremely common predisposing causes in infants and children, the secondary bronchopneumonia causing a higher mortality than the original affection.
4. DEBILITATING AND CHRONIC DISEASES IN OLD AGE.—Especially nephritis, cardiac lesions, and arteriosclerosis.
5. TUBERCULOSIS.—A very common cause.

Aspiration or Deglutition Pneumonia.—When matter containing organisms enters healthy bronchi, an intense bronchopneumonia occurs, so severe that suppuration or gangrene may follow. The entry may be due to:—

(1) LOSS OF THE LARYNGEAL SENSITIVENESS, as in operations under anæsthesia about the nose and mouth, with tracheotomy, in cancer of the larynx and œsophagus, in coma or uræmia, and in various nervous diseases: particles of food or drink pass the larynx and reach the bronchioles.

(2) PASSAGE OF MATTER from diseased portions of lung into healthy bronchioles: may occur in bronchiectasis, hæmoptysis, empyema ruptured into lung, abscess of lung, etc.

SEPTIC EMBOLUS of the pulmonary vessels is a special method by which organisms may reach the bronchioles.

Age. The conditions usually associated with bronchopneumonia at different ages vary greatly. The following is a summary:—

INFANTS.—Under 2 years. Primary bronchopneumonia.

CHILDREN.—Over 2 years (especially 2 to 5 years). Acute specific fevers; rickets; diarrhœa.

ADULTS (uncommon). Aspiration pneumonia; influenza.

OLD AGE.—Debilitating and chronic diseases.

TUBERCULOSIS AT ANY AGE.

Ether Pneumonia may also be lobular.

Season.—Most common in winter and spring.

MORBID ANATOMY.

Both lungs are affected in at least 60 per cent of cases. The condition of lungs post mortem varies greatly. The essential pathological change is a bronchiolitis, the inflammation spreads to the alveoli, and results in proliferation and desquamation of the epithelial cells lining the wall. The macroscopic appearances depend mainly on the extent to which this alveolar change has progressed.

Three groups may be described which correspond to the stages most often seen: (1) Group with *acute bronchiolitis*; (2) Group with *disseminated bronchopneumonia*; (3) *Pseudo-lobar* form.

1. **Acute Bronchiolitis.**—Most commonly seen in severe cases which have died in two or three days. Affection of alveoli insufficient to cause visible consolidation. In early cases macroscopically resembles bronchitis: histologically some alveoli found to be affected. On section, congestion and œdema: crepitant

Bronchopneumonia—Morbid Anatomy, *continued.*

mucopus in bronchi. In cases somewhat later, on section lung has mottled appearance due to minute areas of collapse, consolidation, emphysema, and normal lung.

2. **Disseminated Bronchopneumonia.**—Common type. Lungs fuller and heavier than usual, but mostly still crepitate.

PLEURAL SURFACE.—Three conditions recognizable, viz.: (a) Depressed purple areas of collapse; (b) Areas of normal lung; and (c) Projecting dark areas of consolidation, over which pleura has lost its polish.

CUT SURFACE.—General dark-red colour. Usually smooth, may be granular. Areas similar to those on pleural surface. Areas of collapse can mostly be inflated through bronchus.

MACROSCOPIC CHARACTERS of an area of consolidation. Area is a group of affected bronchioles and the related alveoli. Size of small pea, and upwards. Projects slightly above surface. Colour, grayish-red. Surrounds a small bronchus, which is inflamed and plugged with mucopus. Lung in neighbourhood is dark-red, smooth, and airless; due to earlier stage of inflammation.

MICROSCOPIC CHARACTERS of an area of consolidation.

BRONCHIOLE.—Lumen filled with plug of epithelial cells and leucocytes. Wall swollen and infiltrated. May be irregular dilatations.

ALVEOLI.—*Proliferation of epithelial cells lining wall*: lumen occupied by swollen cells already desquamated, and by leucocytes: fibrin scanty or absent: red cells rare. Walls infiltrated with leucocytes, and contain distended capillaries. Changes most marked in alveoli close to affected bronchioles.

3. **Pseudo-lobar Form.**—Areas of consolidation extensive and coalescent. Intervening areas of congestion usually prevent uniform appearance. Macroscopically may be indistinguishable from true lobar pneumonia, but histologically resembles previous group.

In Aspiration Pneumonia, extensive infiltration with leucocytes occurs throughout affected areas.

BACTERIOLOGY.

No specific organism. *In primary bronchopneumonia*, the bacteriology is probably identical with that of lobar pneumonia, i.e., most commonly due to pneumococci alone; other organisms may be streptococci and staphylococci, usually in association with pneumococci. *In secondary cases*, the infection is usually mixed, two or more organisms being present, of which pneumococcus is commonly one. Common organisms are streptococci, staphylococci, and the influenza bacillus: less common are *Micrococcus catarrhalis*, diphtheria and typhoid bacilli, and Friedländer's pneumobacillus. Occasionally such organisms as *B. pyocyaneus* and *Micrococcus tetragenus*, practically confined to aspiration and septic cases.

SYMPTOMS AND CLINICAL COURSE.

Primary Bronchopneumonia.—This variety, in its onset and symptoms, physical signs, diagnosis, prognosis, and treatment,

may be regarded as identical with lobar pneumonia occurring at a similar age. The distinction depends on morbid anatomy and histology, and the diagnosis is rarely made definitely during life. Mortality is low. This form will not be referred to again.

Secondary Bronchopneumonia.—There is no distinctive clinical course, and symptoms and signs are less definite than in lobar pneumonia.

NATURE OF ONSET.—During convalescence and while suffering from a predisposing cause, commences as bronchitis, and symptoms pass into those of bronchopneumonia, usually slowly but rarely suddenly.

At first slight indisposition. Then actual onset shown by symptoms: *pyrexia, cough, rapid respiration and pulse, and fine râles* on auscultation.

TEMPERATURE.—Usually 102° to 104° F. Generally marked daily variations, 3° F. or more. Never falls by crisis. Hyperpyrexia is bad sign. In severe cases pyrexia may be slight.

COUGH.—Frequent: usually feeble: vigorous cough a good sign.

RESPIRATION.—Rapid, often 60 or more; increases in proportion to extent of lung affected. May be jerky. Pause after expiration common. Retraction of lower ribs and sternum during inspiration points to deficient lung expansion, and is a serious sign.

PULSE.—Rapid, usually small, but may be full at onset.

CYANOSIS.—In severe cases. Always a serious sign. First seen on lips. In grave cases pallor follows.

The above symptoms are the most characteristic and important. Other symptoms are:—

SKIN.—Dry or moist, but rarely pungent.

SPUTUM.—Young children swallow sputum. In old patients, scanty thin mucus, or mucopus.

HERPES.—Not common.

APPETITE.—Impaired. Thirst may be great.

NERVOUS SYMPTOMS.—Marked only in grave cases.

PHYSICAL SIGNS.

Vary greatly. Diagnosis mainly by auscultation.

AT ONSET.—Signs of capillary bronchitis and congestion, viz., percussion note resonant, fine râles, breath-sounds feeble.

LATER.—Râles louder, breath-sounds harsh, vocal resonance louder. On percussion, impaired resonance may be recognized, but definite dullness is rare, and often there is no change.

Death often occurs without these later signs, but extensive areas of consolidation may be found.

Progress of Severe Case.—Asphyxia and toxæmia develop. Anxious expression. Cyanosis, then lividity. Cough diminishes as toxæmia increases. Râles widespread as tubes fill with secretion. Patient becomes restless and sleepless. Inspiratory retraction of ribs marked. Right ventricle dilates. Death occurs.

Bronchopneumonia, *continued.*

Terminations.—Primary and secondary cases end almost invariably in resolution or death.

Other terminations are: *Fibrosis*, leading to chronic bronchopneumonia. Common in tuberculous form, rare in others. *Suppuration*, or gangrene: the common termination of aspiration pneumonia. Very rare in others. Mortality very high.

Cause of Death.—May be (1) Asphyxia and toxæmia; (2) Heart failure; (3) Exhaustion in protracted cases.

DIAGNOSIS.

From the following three conditions diagnosis may be difficult:—

1. ACUTE BRONCHITIS.—At onset diagnosis may be impossible. High temperature, severe constitutional disturbance, and localized bronchitis occurring in children are usually due to bronchopneumonia, simple or tuberculous.
2. LOBAR PNEUMONIA.—Diagnosis is difficult when large areas of bronchopneumonia are confluent (pseudo-lobar form).
   PRIMARY BRONCHOPNEUMONIA. Most common in children under 2 years, while lobar pneumonia is more common after 2 years of age. Diagnosis is of little importance.
   SECONDARY BRONCHOPNEUMONIA.—Special differences: *Child previously in ill-health, onset insidious, affection bilateral.*
3. TUBERCULOUS BRONCHOPNEUMONIA (q.v.). Diagnosis usually possible only by duration: suspected after four weeks. May be suggested by affection of apices, by signs of caseation, and by wasting, but not with certainty. Tubercle bacilli occasionally found in vomit in children, due to swallowed sputum, and very rarely in the fæces.

Cerebral symptoms occasionally suggest meningitis.

PROGNOSIS.

In adults, mortality is very high in *aspiration pneumonia*, and when intercurrent in chronic diseases.

In children, mortality low in *primary form.*

Secondary Bronchopneumonia in Children.—

MORTALITY.—Under 5 years 30 to 50 per cent; in private practice 10 to 20 per cent. Prognosis varies with:—

AGE.—Mortality greatest under 1 year; it decreases steadily with age.

PREVIOUS CONDITION.—With rickets or after acute specific fevers more severe than following bronchitis. Second attack worse than first if interval is short. Thin children do better than fat ones.

TEMPERATURE.—Temperature over 105°, or high and irregular, or low with extensive lung signs, are all unfavourable. Best sign is steady high temperature, 102·5° to 104°.

In a given case prognosis depends on *temperature, cyanosis, extent of lung involved*, nervous symptoms, and state of digestive organs.

In protracted cases, vomiting and gastric disturbance are serious.

No case is ever hopeless.

TREATMENT.

(*See also* LOBAR PNEUMONIA.)

PROPHYLAXIS.—Of great importance. In predisposing conditions, especially measles and whooping-cough in children, great care should be taken to prevent chills.

GENERAL MANAGEMENT.—Confinement to bed, but infants may be nursed. Position changed frequently to assist emptying tubes. Jacket of Gamgee next chest. Room well ventilated, but no draughts. Steam kettle.

DIET.—Milk and milk-foods in plenty. At regular intervals (about 2 hours). Water freely by mouth, and rectal salines if needed.

ALCOHOL.—Extremely valuable, preferably as brandy. To an infant one ounce daily. Always in severe cases.

BOWELS.—Castor oil or calomel.

PYREXIA.—Antipyretics should never be used. Above 105° reduce by hydrotherapy: tepid baths to infants: cold sponging to children most convenient. With asthenic low temperatures attempt to increase warmth: wrap limbs in cotton-wool: hot bottles: hot baths: mustard baths.

RESPIRATORY SYMPTOMS (for prescriptions, *see* ACUTE BRONCHITIS).—In early stage of dry cough give expectorants. Cease when cough becomes loose, as expectorants upset the stomach, and also aid accumulation of secretion in tubes. Inhalations of tinct. benzoin. co. (ʒj to 1 pint) often loosen cough. If robust patient, emetic often effective. When secretion loose, give belladonna. If cough becomes chronic, heroin may be given but an effective cough must not be stopped.

CIRCULATORY SYSTEM.—Treatment of cardiac failure as in lobar pneumonia. In old persons, give stimulants freely; press food, and avoid cold.

CONVALESCENCE. Treatment of great importance. Fresh air, tonics, and full diet. Chills to be avoided.

CHAPTER VI.

CEREBROSPINAL FEVER.*

(*Cerebrospinal Meningitis. Spotted Fever.* (In infants) *Posterior Basal Meningitis.*)

An acute infectious disease, occurring sporadically and in epidemics, caused by the meningococcus, and characterized pathologically by purulent inflammation of the meninges of the brain and cord.

History.—The history previous to 1805 is unknown: possibly confused with typhus. The disease occurs in Central Africa, and may have been imported by Napoleon's army from Egypt.

* *See* Rolleston's "Lumleian Lectures," *Lancet*, 1919.

Cerebrospinal Fever—History, *continued.*

WEICHSELBAUM, 1887, described the *meningococcus.*

STILL, 1898, discovered a diplococcus in posterior basic meningitis.

Recent extensive epidemics: New York, 1904; Glasgow and Belfast, 1907. During the European War, a large increase occurred, initially among the troops; said by some to have been introduced from Canada, or a virulent strain imported thence.

Etiology.—

AGE.—Incidence greatest up to 5 years, in normal circumstances.

SEASON.—Highest in first half of year: attributed to confinement in dwellings and prevalence of colds and coughs.

OVERCROWDING.—The 'carrier rate' among soldiers increases as distance between bunks is decreased.

FATIGUE.—Increases the liability. These two factors account for occurrence amongst soldiers.

RELATION TO OTHER DISEASES.—Nasopharyngeal catarrh and coughs probably aid spread of cocci.

Mode of Infection.—

CARRIERS.—Epidemics are characterized by the irregularity of spread, cases apparently being unconnected. Infection is due to carriers with meningococci in the nasopharynx or accessory sinuses, spread resulting from coughing, etc. Susceptibility is very low, few carriers contracting the disease. In a healthy population, 5 per cent may be carriers: when 20 per cent are carriers, cases of disease begin to occur (Glover). Infection is practically always from a carrier, direct infection from a patient suffering from the disease being very rarely proved.

PATH OF INVASION.—Nasopharynx is infected initially. Theories of the path to the meninges are:—

1. DIRECT TO THE MENINGES BY LYMPHATICS.—Pus is sometimes found in the sphenoidal sinuses (Embleton), and possibly spread may be direct by lymphatics.
2. INVASION OF THE BLOOD.—Producing a *septicæmia with subsequent localization in the meninges.* A septicæmic stage certainly occurs, when, for a short period, meningococci may be isolated by blood cultures. Premeningitic symptoms are also recognizable. This is probable path.

Bacteriology.—

Diplococcus intracellularis meningitidis, or *Meningococcus*, discovered by Weichselbaum in 1887.

MORPHOLOGY.—Mainly in pairs. In cerebrospinal fluid and pus most, but not all the organisms are within the leucocytes (intracellular). Shape either round or flattened. Gram-negative. This closely resembles gonococcus.

CULTURES.—Grow most readily on Gordon's 'trypagar'; large colonies, somewhat opaque. Less readily on ascitic agar. On ordinary agar growth more delicate and often fails. Cultures die readily, and subcultures are necessary every few days.

Involution forms are common in cultures, cocci being swollen and staining badly.

GORDON'S TYPES OF MENINGOCOCCI.—By agglutination with antisera, prepared by inoculating animals with various strains, Gordon has separated 4 types, I, II, III, and IV. Types I and II occur with about equal frequency, forming 90 per cent of all strains: Type IV is very rare. Types I and III are akin to some extent, and classed as Type *A* by certain authorities: similarly Types II and IV have been classed as Type *B*. Type *B* is also known as parameningococcus and Type *A* as meningococcus.

PRESENCE AND ISOLATION OF MENINGOCOCCUS.—
1. Nasopharynx and accessory sinuses in 'carriers'.
2. Blood in early stage of disease. Isolated in about 25 per cent.
3. Cerebrospinal fluid during disease.

Rarely isolated from nasopharynx during disease.

Morbid Anatomy.—General characteristic is a suppurative inflammation of pia-arachnoid, especially at base of brain. In very acute cases (meningococcal septicæmia) condition of hyperæmia only may be present.

CEREBRAL MENINGES AND BRAIN.—Pia-arachnoid injected, and purulent exudate in subarachnoid spaces, especially at base. On cortex often much lymph, especially in larger depressions. Brain substance soft and pink; may be foci of hæmorrhages. Ventricles distended with fluid or even with pus. Microscopically, infiltration along vessels and other channels, and may be foci of encephalitis.

SPINAL CORD.—Always affected, especially posterior surface, and in dorsal and lumbar regions. Pus may surround all the cord, and even nerve roots.

In more chronic cases *meninges* are thickened and remains of ex date present. Cranial nerves usually involved. *Ventricles* may be greatly distended with clear or turbid fluid, and foramen of Magendie closed.

OTHER ORGANS.—Usually these show little change. Spleen occasionally enlarged. May be terminal pneumonia.

Symptoms.—

INCUBATION PERIOD.—From 1 to 4 or 5 days.

MODES OF ONSET.—(1) Ordinary type: Sudden onset. Condition becomes progressively worse, suggesting cerebrospinal meningitis in 24 hours. (2) *Fulminating type*: Abrupt onset. May be mania. Progress very rapid. Comatose within few hours.

Ordinary Form.—*Onset*: Sudden, with headache, vomiting, pyrexia, and, in children, convulsions. Stiffness of neck, head retraction, and general irritability develop. General condition of irritation of the nervous system and increased intracranial pressure. The occurrence of an initial nasopharyngeal catarrh is doubtful. Temporary improvement occasionally follows onset.

MOTOR SYMPTOMS.—
1. HEAD RETRACTION.—May be extreme. In infants, opisthotonos.
2. RIGIDITY.—(i) Kernig's sign, rarely absent. (ii) Brudzinski's

Cerebrospinal Fever—Symptoms, *continued.*

'neck sign': if the head is flexed by the hand, with the patient lying on his back, flexion of the knees and thighs occurs (a valuable sign of meningitis). (ii) Brudzinski's 'leg sign': if one leg be flexed, flexion also occurs in the opposite leg.

3. REFLEXES.—Deep reflexes (knee-jerks) usually increased. Babinski's sign in about 10 per cent.
4. SPASMS.—Commence as twitching, increasing to clonic or tonic spasms. Tremor common.
5. OCULAR SYMPTOMS.—(i) Pupils: Usually dilated, from irritation of sympathetic; may be contracted, in severe forms. Inequality and sluggish reaction common. Hippus not infrequent. (ii) Strabismus: In about 20 per cent. (iii) Optic neuritis. Not common; about 10 per cent. Photophobia, conjunctivitis, ptosis, nystagmus occasionally.

SENSORY SYMPTOMS.—Headache often very severe, especially occipital. Pain may extend along spine and limbs. General hyperæsthesia may occur.

MENTAL SYMPTOMS.—At onset restlessness or delirium, later stupor and coma.

VOMITING. Of the cerebral type, very frequent at onset, may continue or subside later.

TEMPERATURE.—Irregular, no typical course, remissions and intermissions common; may rise to 105° or over; about 103° usual.

PULSE.—Slow in relation to temperature, may be irregular.

RESPIRATION.—Towards termination may be Cheyne-Stokes. Only increased with pulmonary complications.

CUTANEOUS SYMPTOMS.—

1. RASH.—Onset early, 1st or 2nd day. Rash is hæmorrhagic, either (*a*) petechial, or (*b*) purpuric (fulminating cases only). Incidence before the war very rare: during the war in 60 per cent.
2. HERPES LABIALIS.—In 25 to 50 per cent. Onset not before 4th or 5th day.

Urticaria and other rashes may occur.

BLOOD.—Polynuclear leucocytosis, 25,000 to 50,000 per c.mm. Leucocytosis may be absent in fulminating cases.

EMACIATION.—Often very rapid.

Other Clinical Types.—

1. FULMINATING FORM.—Abrupt onset: headache, vomiting, collapse: purpuric rash common. Temperature high or low. Rapid coma. Death in a few hours. Cerebrospinal fluid may be clear and contain no cocci. Hæmorrhage in medulla of suprarenals frequent (the medulla is of nervous origin). Abdominal symptoms may occur. Probably two types: (*a*) Acute meningococcal septicæmia; (*b*) Acute infection of meninges.
2. MILD AND ABORTIVE FORMS.—Symptoms mild or subsiding in a few days.
3. CHRONIC FORMS.—Recrudescences may occur over many months. Other chronic forms are associated with closure by

meningitis of the foramina of Magendie and Luschka: the ventricles are distended either with pus, turbid fluid, or clear fluid, constituting 'closed ventricular meningitis' or hydrocephalus. Common in posterior basic meningitis.

POSTERIOR BASIC MENINGITIS.—Cerebrospinal meningitis in infants. Commonest form of meningitis under age of one year. Note: (1) Head retraction and opisthotonos marked; (2) Rash rare; (3) Loss of vision without optic neuritis common; (4) Often very chronic; (5) Sequelæ usual in non-fatal cases: deafness and hence deaf-mutism, blindness, mental deficiency, general spasticity of extremities (hydrocephalus)

PROGNOSIS.—Death: 50 per cent. Complete recovery 15 per cent. Various sequelæ 35 per cent.

Complications and Sequelæ.—

NERVOUS SYSTEM.—Facial paralysis, hemiplegia, and paraplegia occur may be functional or organic. In the chronic forms and hydrocephalus, attacks occur with headache, vomiting, mental dullness, and dilated pupils.

CIRCULATORY SYSTEM. Pericarditis: rare, not always fatal, often latent and found at autopsy. Endocarditis: rare.

PULMONARY SYSTEM. Pneumonia or pleurisy is rare; occasionally a terminal complication.

ARTHRITIS OR SYNOVITIS. Occurs in 5 to 10 per cent a previous hæmorrhagic rash is almost invariable. Suppuration is rare and prognosis good.

OTITIS MEDIA OR LABYRINTHITIS. Occasionally results from extension along auditory nerve.

EPIDIDYMITIS AND ORCHITIS. Rare.

Cerebrospinal Fluid.—

CHARACTERS.—(1) *Amount increased* and under abnormal pressure; (2) Fluid *turbid* or purulent; (3) *Protein increased*; (4) *Polynuclear leucocytes* present in deposit; (5) *Meningococci* present, intra- and extracellular but may be absent even with turbid fluid; (6) *Dextrose absent*: the cause of this is doubtful, possibly fermented by meningococci, or due to action of leucocytes. The fluid may be clear for the first 24 hours. In later stages, with closure of foramen of Magendie by meningitis, amount of fluid may be scanty. Mixed infections occasionally occur, usually pneumococci.

Diagnosis.—

CLINICAL CHARACTERISTICS.—At onset headache, vomiting, pyrexia, stiffness of neck, and delirium: evelopment of head retraction.

SPECIAL METHODS.—(1) Lumbar puncture: pathognomonic except occasionally in first 24 hours. (2) Blood-count and blood-culture: of less value.

DIAGNOSIS FROM.—(1) Other conditions which produce meningeal symptoms: typhoid fever, pneumonia, influenza, otitis media. (2) Other causes of meningitis: tuberculous, or rarely

Cerebrospinal Fever—Diagnosis, *continued.*

pneumococcal. (3) Acute poliomyelitis. (4) Encephalitis lethargica. (5) Typhus, and rarely other conditions with purpuric eruptions.

Prognosis.—Bad in: (1) Infancy and over 40 years of age; (2) Fulminating forms; (3) Purpuric rashes; (4) Pulmonary complications. Condition of cerebrospinal fluid of comparatively little value unless cocci very numerous: pus may disappear rapidly. Temperature of little prognostic value.

DURATION.—Very variable. Death frequently towards end of first week, but may occur later.

CONVALESCENCE.—Many months.

RECRUDESCENCES.—Common before recovery, but true relapse after complete recovery is rare.

MORTALITY.—Without serum 50 to 70 per cent: with efficient serum treatment should not exceed 30 per cent.

Treatment.—

LUMBAR PUNCTURE.—Should be performed at once, even in doubtful cases, for diagnosis and for introduction of serum. Withdrawal of fluid relieves headaches and reduces intracranial pressure, but must be combined with serum treatment.

SERUM TREATMENT.—Should never be omitted even in doubtful cases.

ESSENTIALS are: (1) Early injection; (2) Serum employed must contain antibodies to the infecting strain.

ANTISERA IN USE include: (i) Flexner's serum, polyvalent, prepared from numerous strains; (ii) Medical Research Council's monotypical sera for each of Gordon's Types (Gordon); (iii) Medical Research Council's pooled serum for Types I and II. Before the type of meningococcus is ascertained, either Flexner or the M.R.C. pooled serum should be used. *Note.*—Type I and II include 90 per cent of all cases.

DOSAGE.—On first 2 days, 30 to 40 c.c. repeated twice. On next 4 days, 30 to 40 c.c. daily: subsequently continue daily until fluid clear and temperature falls. Repeat if recrudescence occurs. At onset, if fluid is clear, an intravenous injection is recommended in addition to above to neutralize the meningococcal septicæmia (200 to 500 c.c.).

TECHNIQUE.—Warm serum to body temperature. Perform lumbar puncture and allow cerebrospinal fluid to drip away. The amount of serum *must never exceed* the amount of fluid removed. The serum is introduced by gravity: the bowl of a syringe being connected to the trocar by a rubber junction; the serum *must not be forced* into the thecal space.

GENERAL HYGIENE AND TREATMENT.—As in tuberculous meningitis. Feeding through nasal tube should be employed without hesitation, as a nutritious diet is of great importance. Local treatment to nasopharynx is of doubtful value.

Hexamine (urotropine): Value is not proved, but drug is harmless: is secreted into cerebrospinal fluid. For adult, gr. x. t.d.s.; for infant, gr. ij.

Prophylaxis.—

1. GENERAL HYGIENE. - Fresh air and sufficient cubic space in barracks, etc.
2. SEARCH FOR CARRIERS.—The elimination of carriers would extinguish the disease, but cannot be carried out completely. Many carriers also appear to be intermittent, regarding results of examination. When a case has appeared, contacts should be examined by swabs of the nasopharynx.

   TREATMENT OF CARRIERS.—At present unsatisfactory. Various forms of local sprays, and steam sprays, have been employed. Also vaccines.

PROPHYLACTIC INOCULATION.—Results have been encouraging, but data insufficient.

Quarantine Period for Contacts.—Seven days.

CHAPTER VII.

INFLUENZA.*

An acute infectious disease especially attacking the respiratory tract, but characterized by the variability of the symptoms, a post-febrile nervous stage, and widespread epidemics. The *B. influenzæ* has been isolated in many epidemics and sporadic cases.

Etiology.—A pandemic occurred in 1889–90. It commenced probably in Turkestan, and spread from East to West, becoming world-wide within 12 months. Epidemics recurred in 189[illegible] and 1892, in the latter year being almost pandemic. In subsequent years, local epidemics have occurred, but on a smaller scale. Propagation is direct from person to person: infectivity is very high and spread very rapid. Epidemics are independent of personal, seasonal, and usual epidemiological factors. One attack in no wise protects, but the progress and cessation of epidemics suggest that a nation may acquire some immunity. The rapidity of spread depends on the shortness of the incubation period, universal susceptibility, and the frequency of mild neglected cases. Man alone is susceptible: all animals, except possibly monkeys, being immune.

Bacteriology.—*B. influenzæ* was discovered by Pfeiffer in 1892, and practically simultaneously by Kitasato and Canon.

MORPHOLOGY.—Minute non-motile bacillus or cocco-bacillus Straight with round ends. Does not form spores. In sputum and body fluids occurs singly and in clumps, both intra- and extracellular. Gram-negative. Stains with all ordinary stains.

CULTURAL CHARACTERISTICS.—Isolated best on *Pfeiffer's blood-agar* (blood spread on agar). Forms transparent colonies. Growth delicate, dies rapidly in subcultures. *No growth on ordinary media*: hæmoglobin is essential. *Pure aerobe.*

* The epidemic of 1918 is referred to at the end of the section.

Influenza—Bacteriology, *continued.*

DISTRIBUTION IN THE TISSUES.—In the respiratory tract, bronchi, bronchioles, and lung. In sputum often in large numbers. In pus from empyemata may be present in pure culture, but streptococci and other organisms often present. Rarely isolated from the blood. Occasionally isolated in meningitis, otitis media, and other lesions following influenza.

RELATION OF B. INFLUENZÆ TO INFLUENZA.—Pfeiffer's bacillus was generally accepted as the cause of the epidemic of 1889 and immediately succeeding years. In subsequent epidemics it was not always found. In certain epidemics, apparently influenzal, other organisms have been present, e.g., *Micrococcus catarrhalis.*

Morbid Anatomy.—In fatal cases, inflammatory changes in the lungs are invariably present, most commonly bronchopneumonia: no specific lesions.

Symptoms.—

INCUBATION PERIOD.—Two to five days.

The symptoms are extraordinarily complex and variable, but certain types can be recognized: (1) General febrile type; (2) Respiratory; (3) Nervous; (4) Gastro intestinal

1. GENERAL FEBRILE TYPE.—Under this heading are described the general features commonly seen in an attack of influenza.

   ONSET ABRUPT.—Often sudden severe vertigo.

   HEADACHE.—Severe. Frontal or very frequently at back of eyeballs. Pain on movements of eyes.

   PAIN IN BACK AND IN BONES.—Often very severe.

   TONGUE.—Furred, and breath offensive.

   CORYZA.—Bronchitis common. Conjunctivitis.

   PROSTRATION RAPID.

   CHILLS.—Especially sensation of 'goose-flesh'. Later on drenching sweats.

   FEVER.—Lasting three to five days. Pulse usually not increased in proportion to temperature.

   PHYSICAL SIGNS.—A few râles at the bases or nothing at all. Spleen occasionally palpable.

   RELAPSES.—Common.

   Acute symptoms usually last about one week.

   The general febrile form may develop into any of following types, or these may dominate the symptoms from onset.

2. RESPIRATORY TYPE.—Respiratory symptoms marked.

   BRONCHITIS.—Sputum usually in very large amounts: may be purulent. Scattered râles in lungs.

   PLEURISY.—Empyema very frequently follows. Streptococcus or pneumococcus usually present, less commonly *B. influenzæ.*

   PNEUMONIA.—Always serious; accounts for nearly all deaths. Practically always lobular.

3. NERVOUS TYPE.—Symptoms variable. May be very severe. Headache, insomnia, delirium, prostration common.

4. GASTRO-INTESTINAL TYPE.—*Rare.* Attack may commence with abdominal pain and profuse diarrhœa; may be

nausea and vomiting. Respiratory symptoms often entirely absent. Jaundice may occur. Spleen may be enlarged.

THE HEART.—May be especially affected. Uncommon, but serious. In acute stage, rapid, irregular pulse. Convalescence, slow, liability to tachycardia and dilatation.

FEVER.—Variable, no typical course, usual duration about five days; may last three weeks.

Complications and Sequelæ.—There is almost invariably depression of physical, and, more especially, mental powers. Frequently also vertigo, palpitations, and vague neuralgia.

NERVOUS SYSTEM.—

Psychical sequelæ of all varieties from depression to suicidal tendencies. Common are insomnia, loss of smell and taste, irritability of temper, also many forms of neuralgia and neuritis. Neurasthenia or melancholia may last for months, or rarely years.

Numerous lesions have been described, e.g., acute polyneuritis, paralyses of all types.

RESPIRATORY SYSTEM.—Pulmonary complications are very important and frequent: *pneumonia*, which may terminate in gangrene; *chronic bronchitis* is common; rarely bronchiectasis.

CIRCULATORY SYSTEM.—Vertigo, palpitations, tachycardia, and cardiac weakness may be persistent. Acute dilatation and sudden death rare. Infective endocarditis, pericarditis, rare.

SUPPURATION.—Local abscesses may form in any site, especially middle ear, antrum of Highmore, and superficially.

Rarer complications are thrombosis of vessels and nephritis.

Diagnosis.—During an epidemic, diagnosis is usually easy. In sporadic cases and small outbreaks, diagnosis frequently made solely by the extreme prostration in the post-febrile stage: often very uncertain. *B. influenzæ* may be found in sputum.

Treatment.—

GENERAL TREATMENT.—Confine to bed until temperature has been normal several days and *no râles are present in lungs.*

DRUGS.—There is no specific, but a course of quinine should be given, or aspirin (gr. xv, t.d.s.).

BOWELS. Commence with calomel (gr. ij) on first night and saline in morning.

INITIAL CORYZA.—Tinct. quin. ammon. ʒj every 4 hours.

HEADACHE.—Phenacetin gr. x.

SEVERE GENERAL PAINS.—Aspirin or sodium salicylate (gr. xx every 4 hours). Dover's powder gr. x.

INSOMNIA.—Paraldehyde ʒj, in whisky.

COUGH.—Heroin or a simple linctus. (*See* ACUTE BRONCHITIS.)

HYPERPYREXIA AND DELIRIUM.—Treat as in typhoid fever.

CARDIAC WEAKNESS.—Alcohol, digitalis, and strychnine.

Localized symptoms in lung and alimentary system need the appropriate treatment.

CONVALESCENCE.—Change of air, good diet, arsenic and quinine; avoid chills.

Influenza, *continued.*

Epidemic of 1918.—

Cases commenced to appear early in the year. In the later spring and summer, the number affected became very large. Clinically, the onset was abrupt, the temperature rising rapidly, often to 104°, the face flushed, and vertigo and headache severe. The duration was short, usually 3 to 5 days, the temperature falling rapidly. Convalescence was rapid. Severe cases of pulmonary symptoms and complications were rare and mortality very low.

In the autumn, a second wave occurred, characterized by greater severity and a relatively large proportion of pulmonary complications, with very high death-rate.

DIFFERENCES FROM PREVIOUS EPIDEMICS.—In 1918 —

1. Young adults, age 20 to 40 years, especially affected, and mortality very high.
2. Pneumonia very frequent cause of all deaths. Cyanosis marked.
3. Symptoms did not exhibit variability of previous epidemics. Complications rare except pneumonia. Nasopharyngeal affections not prominent.

BACTERIOLOGY.—*B. influenzæ* nearly always present, but its relationship to the epidemic and symptoms is hotly disputed. Streptococci invariable in lungs at autopsy and an undoubted factor. Presence of 'filter passers' still in dispute.

MORBID ANATOMY OF LUNGS.—Lesions bilateral, mainly at bases: always bronchopneumonic, slate-blue areas of consolidation: hæmorrhages numerous.

TREATMENT.—In pulmonary forms as for severe lobar pneumonia. Alcohol freely and other cardiac stimulants.

PROPHYLACTIC INJECTIONS.—Vaccine containing per c.c. *B. influenzæ* 500 million, pneumococci 1000 million, streptococci 100 million. Two injections, 0·5 and 1 c.c., at intervals of a week. Results promising.

Quarantine Period.—Five days is sufficient.

CHAPTER VIII.

WHOOPING-COUGH.

(*Pertussis.*)

A specific infectious disease characterized by catarrh of the respiratory tract and paroxysms of coughing terminating in a 'whoop'.

Etiology.—Sporadic cases common. Epidemics frequent. Temperate climates especially affected.

SEASON.—Most prevalent in winter and spring. Maximum in March, minimum in September.

AGE.—*Usually under six years*, but no age immune. Not uncommon in infants. In old people usually severe.

Females in excess of males. *One attack usually protects.* Association with measles very common. *Susceptibility* great but not universal.

Quarantine Period for Contacts.—Sixteen days.

Bacteriology.—Bordet-Gengou, in 1906, described the *Bacillus pertussis*. Isolated on special blood-agar media from tenacious mucus voided at end of paroxysms. A small, Gram-negative, non-sporing bacillus resembling *B. influenzæ*. Complement-deviation occurs with the serum of convalescents. Agglutinins may also be present. Intratracheal inoculation in monkeys produces cough and pyrexia. The bacillus is generally accepted as the cause of whooping-cough, but proof is not yet absolute.

Morbid Anatomy.—No specific changes. Lesions post mortem usually those of some fatal complication. In uncomplicated fatal cases, areas of collapse and emphysema; enlarged tracheal and bronchial glands.

Mode of Infection. *Direct contagion* from the sputum. A very short exposure may be sufficient. The cough can probably project particles to some distance, but with precautions the tendency to spread, e.g., in a ward, is considerably less than with measles. Transmission by fomites, infected clothes, etc., is definitely proved, but probably rare. Cats and dogs are subject to whooping-cough.

Symptoms.—Divided into catarrhal and paroxysmal stages.

INCUBATION PERIOD.—Usually one week; may be 4 to 14 days.

1. CATARRHAL STAGE.—*Onset insidious.* Commences with slight malaise, coryza and cough: not severe, but cough out of proportion to catarrh. Slight bronchitis in lungs. *Pyrexia* slight and intermittent. Some gastric disturbance.

   COUGH.—Becomes more frequent and paroxysmal, especially at night: inspiratory spasms develop; finally characteristic whoop starts. In some cases, whooping occurs almost at once: in others greatly delayed, or not at all.

2. PAROXYSMAL STAGE.—Dated from first whoop. Coryza has previously subsided. Pyrexia slight or absent.

   COUGH.—Course of events in typical paroxysm: (i) *Long inspiration* (often absent), followed at once by (ii) *Series of short expiratory barks.* Thorax fixed, no air enters, face becomes congested. When apparently suffocating, (iii) *Inspiratory whoop.* Congested appearance rapidly passes, but child is exhausted. *Vomiting frequently follows* even in catarrhal stage, and suggests diagnosis. Cycle may recur several times in succession. May be small amount of tenacious mucus at end of paroxysm. Number of *paroxysms* up to 40 a day: *distinctly more frequent at night.* Child becomes aware of oncoming paroxysm, makes attempts to suppress it, and becomes terrified. After attack sleeps, or older children complain of headache. Violent sneezing may precede or follow paroxysm.

   FACE.—Often bloated from the constant congestion: swelling most marked about the eyes: often suggestive.

   SUBLINGUAL ULCER.—Occasionally present: confined to infants with only two lower central incisors erupted. Never before paroxysmal stage.

Whooping-cough—Symptoms, *continued.*

PAROXYSMS.—Usually spontaneous: may be excited by close atmosphere, crying, eating, excitement, or recumbent position. In infants, whoop usually absent: in aged, an occasional whoop.

Physical Signs.—In lungs: very slight. During expiratory coughs, resonance may be defective and a few râles at bases. Pulse becomes very rapid.

Blood Changes.—The total of leucocytes is increased, but more characteristic is the increased percentage of lymphocytes; this may rise to 80 per cent.

Progress.—Paroxysms become less frequent and less severe, and the whoop gradually disappears.

Duration.—Very variable. *Catarrhal stage*, about one week, from three days to two weeks. *Paroxysmal stage*, four weeks and upwards. *Total duration*, usually six to eight weeks, but may be greatly prolonged. *Adenoids* may cause prolongation.

PERIOD OF ISOLATION.—At least six weeks: until whoop has been absent for two weeks and until paroxysms *cease to be frequent*. After cessation of 'whoop', cough may remain paroxysmal; it is unnecessary to regard this stage as infectious, if the foregoing provisos are fulfilled.

Complications.—Important.

1. PULMONARY COMPLICATIONS.—Cause nearly all fatalities.
   - CAPILLARY BRONCHITIS AND BRONCHOPNEUMONIA.—Child remains ill between the paroxysms. Whoop may disappear. Sometimes is tuberculous. Lobar pneumonia rare.
   - COLLAPSE OF LUNGS.—Especially in rickety infants. Due to blockage of air-spaces by tenacious secretion.
   - EMPHYSEMA.—May develop.

   Inspiratory whoop may not occur, and suffocation results, but very rare.
2. VOMITING AND EMACIATION.—The normal vomiting may become excessive.
3. ENLARGEMENT OF BRONCHIAL GLANDS.—Very frequent.
4. CONVULSIONS.—Common in infants. Usually fatal.

HÆMORRHAGES.—Extreme venous congestion may lead to various hæmorrhages, e.g., petechial rashes, conjunctival ecchymosis. Rarely meningeal hæmorrhage, fatal.

ALBUMINURIA occasionally, but nephritis very rare.

PARALYSES AND PERIPHERAL NEURITIS.—Very rare.

Sequelæ.—

TUBERCULOSIS, pulmonary or glandular, not uncommon sequel.

CHRONIC PULMONARY DISEASES, e.g., bronchitis, also emphysema. Ordinary coughs may subsequently tend to be paroxysmal; and in adults asthma may develop.

DEFORMITIES OF THE THORAX, e.g., 'pigeon-breast', may follow a prolonged attack.

CARDIAC WEAKNESS may result from the repeated strain.

RELAPSES AND SECOND ATTACKS.—Rare.

Diagnosis.—

CATARRHAL STAGE.—Often very difficult. Note: (i) Cough out of proportion to signs in lungs, (ii) Cough becoming paroxysmal, especially at night; (iii) Cough accompanied by vomiting.

PAROXYSMAL STAGE.—Typical cases easy, but in young infants whoop may be absent throughout.

BLOOD.—Increase in total leucocytes and in percentage of lymphocytes. Often valuable.

DIAGNOSIS FROM.—

MEASLES.—Koplik's spots, occurrence of rash.

ENLARGED BRONCHIAL GLANDS AND ADENOIDS.—May cause convulsive cough: no whoop, vomiting rare.

LARYNGISMUS STRIDULUS.—No whoop, no bronchial catarrh.

Cause of the Whoop. Uncertain. Has been ascribed to laryngeal spasm from local irritation of larynx by mucus (doubtful). Possibly specific irritation of vagus.

Prognosis.—MORTALITY: varies greatly with age: under 1 year high, and under 3 years considerable; over 5 years, less than 1 per cent; severe in the aged. CONVULSIONS: high mortality. BRONCHOPNEUMONIA accounts for most deaths. Tuberculosis and chronic pulmonary diseases not infrequently develop later.

Treatment.—

1. GENERAL TREATMENT.—Preferably isolated in two rooms. Temperature maintained at 60 to 63°. *Fresh air essential.* Confine to bed during catarrhal stage or pyrexia. Cotton-wool jacket. Support child during paroxysm. Abdominal binder comforting. Confine to rooms for three weeks at least, not necessarily in bed.
2. DIET.—Milk and milk foods and meat-juice: small and frequent meals. Food well administered immediately after a paroxysm.
3. DRUGS.—Eucalyptus oil should be sprinkled on the bed-clothes, or may be evaporated in a saucer over a spirit lamp, or from a steam kettle (ʒj to a pint). The nose and throat may be sprayed with a simple antiseptic, e.g., listerine and glycerin, *unless this causes paroxysms*, but should not be attempted in young children. Rub chest with a stimulating liniment (lin. camphor. for infants, lin camphor. ammon. for children).
   (1) CATARRHAL STAGE.—Expectorants, especially ipecacuanha, as in bronchitis.
   (2) PAROXYSMAL STAGE.—Give sedatives. Bromoform (♏ss to ij on sugar) and potassium bromide are suitable. Belladonna, a traditional remedy, should be given in full doses, combined with sedatives: dose ♏j, t.d.s., at age of one year. Paregoric (tinct. opium camphorat. co.) is valuable, especially a dose at night. Heroin is perhaps the best drug, given as a linctus: for prescription, *see* BRONCHITIS. Adrenalin: good results have been recorded. If paroxysms are very severe, administer chloroform.

LINGUAL ULCER.—Bathe with weak myrrh and borax.

As paroxysmal stage passes, quinine is useful.

Vaccines and intratracheal injections are under trial.

Whooping-cough—Treatment, *continued.*

CONVALESCENCE.—Great care should be taken to avoid chill, owing to risk of tuberculosis and pulmonary diseases, but fresh air is of greatest importance, and child need not be confined to the house until all paroxysms have ceased. Give cod-liver oil and malt and iron tonics, e.g., syr. ferri phosphatis co.

If attack is prolonged, try change of climate: examine for adenoids, and remove if present.

Measles is not uncommon during convalescence.

CHAPTER IX.

GONOCOCCUS INFECTIONS.

An infection by the gonococcus, with a primary lesion usually in the urethra, various lesions in the genital tract due to direct extension, and a liability to systemic infection. The lesions in the genital tract are not described here.

Etiology.—In new-born: occurs as ophthalmia neonatorum, due to vaginal infection of conjunctiva. Amenable to early treatment, but neglected cases are a common cause of blindness. In infants and children: as vulvovaginitis from accidental infections by sponges, etc. In adults: spreads by sexual intercourse with infected individuals.

Bacteriology.—Gonococcus was isolated by Neisser in 1889.

PRINCIPAL CHARACTERISTICS ARE: (i) Diplococcus, bean-shaped with flat sides almost in apposition; (ii) Gram-negative, but stains with ordinary stains; (iii) In pus and body-fluids mainly intracellular; (iv) Characteristically present only in a few cells amongst many, such cells each containing a large number of cocci; (v) Grows best on blood-agar and media containing serum or blood. Growth is delicate: does not grow on agar and many ordinary media: cultures die rapidly, especially initial cultures. Life outside body-tissues and media is very short.

Clinical Conditions in Adults.—(i) PRIMARY LESION in man is a urethritis, in woman a cervicitis and urethritis. (ii) DIRECT SPREAD may occur to prostate, epididymis, Fallopian tubes, ovaries, and even through this route to peritoneum. In males gonorrhœal peritonitis is extremely rare. Proctitis is not uncommon in females. *Conjunctivitis* is not very common. (iii) SYSTEMIC INFECTIONS occur in a small proportion of cases. Although gonococcus is not commonly isolated, local lesions are due probably to presence of organisms, and not to toxins absorbed from a distant focus. Systemic infections may be:—

1. SEPTICÆMIA.—Rare: organism sometimes isolated from blood. Clinical types: (i) General septicæmia, condition may resemble typhoid; (ii) Pyæmic abscesses; (iii) Gonorrhœal

puerperal septicæmia; (iv) Infective endocarditis and pericarditis: very rare. Fatal termination is rare, except in infective endocarditis.

GONORRHŒAL ARTHRITIS.

TIME OF ONSET.—Usually within few weeks or months of initial urethritis, but may be later when gleet is chronic. In rare cases follows the vulvovaginitis of infants and ophthalmia neonatorum.

SEX.—More common in males.

MORBID ANATOMY.—Changes mainly in *periarticular tissues*, œdematous swelling and infiltration. Synovial membrane hyperæmic and joint may contain increased and turbid fluid. polynuclear cells often numerous but suppuration rare. In chronic stages, peri-articular tissues thickened, but bony changes rare. Gonococci may be present in fluid: usually absent. Mixed infections very rare

JOINTS AFFECTED.—*Knee* especially frequent. *Usually more than one joint. Large joints most common. Temporomaxillary* and, rarely, sternoclavicular and sacro-iliac joints may be affected (These escape in acute rheumatism.)

PHYSICAL SIGNS. - Variable. May be stiffness and vague pain, without swelling, or with synovial effusion. More typically, red, hot, and tender, with *peri-articular swelling*, with or without much effusion. *Suppuration rare.* Mixed infections very rare.

CLINICAL COURSE. Duration of joint affection several weeks. as one clears, another often becomes affected. Often very obstinate. Rapid shifting, as in acute rheumatism does not occur.

COMPLICATIONS, SEQUELÆ, AND VARIATIONS IN LESIONS. —The peri-articular tissues are specially affected, and spread may occur along the tendons. Gonorrhœa tends also to attack fibrous tissue. The following are important:—

1. *Fibrous Adhesions.* Commonly form round affected joint, in absence of suitable treatment. Cause contractions and limitation of movement. Bony ankylosis rare.
2. *Flat-foot.*—Common sequel when foot and ankle affected. Caused by yielding of ligaments and plantar fasciæ.
3. *Tenosynovitis.* — Joint may be unaffected. Tendo Achillis most frequent site.
4. *Bursitis.*
5. *Painful Heels.*—Pain in os calcis on walking: probably periostitis of os calcis, or certain plantar fasciæ affected.

CONDITIONS SOMETIMES GONORRHŒAL.—

Sciatica.—May be true sciatic neuritis or neuralgic pains Neuritis of other nerves occurs occasionally.

Spondylitis Deformans. (q.v.).

Acute Myositis. — Painful muscles: usually, but not always, near an affected joint.

Gonococcus Infections—Arthritis, *continued.*

DIAGNOSIS.—Initial lesion usually makes diagnosis easy in males. Important symptoms are involvement of unusual joints, periarticular thickening, obstinate nature, slight fever, and uselessness of salicylates.

Diagnosis especially from acute rheumatic fever, arthritis deformans, and gout.

PROGNOSIS.—Condition obstinate, but prognosis good. Recurrences frequent.

TREATMENT.—

Primary Lesion.—This must be thoroughly treated.

Local Treatment.—Complete rest on splint, but with *massage and passive movements* from early stage in order to prevent adhesions. Paint with iodine. Aspirate if joint very distended. If suppuration occurs, incise and drain. When adhesions are present, break down by careful movements under anæsthesia.

Drugs and antiserum are useless.

Vaccine treatment should be tried in obstinate cases. Dose may commence with 5 million, weekly injections with increasing amounts. Very large injections (500 million) have been given without ill effects.

CHAPTER X.

DYSENTERY.

Dysentery is characterized clinically by: (1) Passage of frequent small stools; (2) Presence of mucus and blood; (3) Abdominal pain and tenesmus. These symptoms constitute dysentery when due to certain specific causes. The symptoms are the result of an *ulcerative colitis*, a condition which may also arise from causes and organisms not at present recognized as dysentery.

Types.—Dysentery is of two main types: (1) BACILLARY, due to certain specific bacilli; (2) AMŒBIC, due to *Entamœba histolytica* (considered here for convenience).

The term dysentery is used now to imply the presence of one of these two groups of organisms, however mild the symptoms may be, and its definition is etiological rather than clinical. Either may cause a simple diarrhœa without the characteristic symptoms. Extensive epidemics are usually bacillary dysentery, and, when the death-rate is high, are generally due to Shiga's bacillus.

I. BACILLARY DYSENTERY.

Bacteriology.—Two principal groups of bacilli: (1) *Shiga* or *Shiga-Kruse.* Isolated by Shiga in 1898. (2) *Flexner.* Numerous strains, at least five, exist in this group, and are identified serologically as V, W, X, Y, Z. Shiga's group is much purer.

MORPHOLOGY.—Non motile, non sporing, Gram negative bacilli resembling coli typhoid group (Motility and flagella have been described in some strains) Grow readily on ordinary media Growth resembles typhoid but is moister and more slimy

CULTURAL CHARACTERS —

LITMUS MILK —Slight initial acidity, then alkaline Never clotted

CARBOHYDRATES —No gas formed by any strain, and all are non lactose fermenters

SHIGA - Acidifies dextrose only

FLEXNER —Acidifies dextrose and mannite some strains, also maltose

INDOLE — Produced by Flexner but not by Shiga

PATHOGENICITY TO ANIMALS —Intraperitoneal injections are pathogenic to guinea pigs, rabbits, and other animals Death occurs in a few days hyperæmia and catarrh of the intestines are present, but the characteristic changes of dysentery are produced only by special methods

Modes of Infection. Resemble enteric, viz, by water, food, flies, and contamination by excreta of infected persons

Morbid Anatomy.- The large intestine is mainly affected The entire colon may be equally involved but frequently the maximum change is in the sigmoid, extending above and below with diminishing severity The ileum is frequently hyperæmic for a varying distance

In acute, rapidly fatal cases, the mucous membrane is hyperæmic dark red, and thickened there is superficial necrosis but usually no ulceration.

In less acute forms, the changes consist of (1) *Thickening of the mucous membrane* may be nearly ½ inch thick most marked on summits of folds, on which in extreme instances polypoid masses may form (2) *Ulceration* commences in the lymphoid follicles, numerous small superficial ulcers forming The edges may be thickened and infiltrated but are never undermined (as occurs in amœbic dysentery) In severe chronic cases the ulceration may affect most of the intestine, a few islets of thickened mucous membrane remaining

Peritoneal adhesions may form

Mesenteric glands are not uncommonly enlarged.

Symptoms.—

INCUBATION PERIOD —May be a few hours only, and probably rarely exceeds three days Occasionally up to eight days.

ONSET —Sudden Characteristic symptoms usually present from the first, the occurrence of a simple diarrhœa at onset being unusual

SYMPTOMS AT ONSET —

FREQUENT SMALL STOOLS.—May be almost continuous.

ABDOMINAL PAIN —Tormina and tenesmus. Between stools there may be little pain.

Bacillary Dysentery—Symptoms, *continued.*

CHARACTER OF STOOLS.—Each motion of small quantity. Mucus at first, then blood and mucus or pure blood. A few initial stools may empty the intestine of fæcal matter.

VOMITING.—Common at onset: may be for one to two days.

HEADACHE.—Usual.

TEMPERATURE.—Variable: high, low, or moderate.

PULSE.—Rapid.

PROGRESS.—Generally rapid, and within one to two days can be divided into (*a*) Severe, (*b*) Moderate.

(*a*) SEVERE FORMS.—Complaints of (i) abdominal pain, (ii) thirst. On examination, dryness and coldness are marked.

Stools. Very numerous. Almost pure blood, with varying amount of mucus. Desire for stool almost continuous.

Skin. Dry and inelastic. A bluish flush on cheeks, of limited area, is common

Extremities cold.

Abdomen. Retracted. Rigidity is unusual. Tenderness often extreme, especially on left side, but on palpation contraction of muscles usually does not occur. Pain preceding and accompanying stools, but may be only slight between motions.

Tongue dry. Fur variable, may be absent.

Pulse rapid and small

Temperature. No characteristic. usually high, 103°, or subnormal

Vomiting not infrequent, and a very serious symptom when occurring at this stage. Also *hiccough*

Muscular pains not uncommon: especially anterior thigh and calves. Also in knees and joints.

Subsequent progress (1) Symptoms more severe. Prostration increases. Discomfort extreme. Incontinence of urine and fæces. Mental wandering common, but mind may remain clear. Progressive failure and death. (2) Slow gradual improvement. Convalescence prolonged Rapid recovery does not occur.

Mortality in severe forms over 50 per cent.

(*b*) MODERATE FORMS.—Not necessarily mild, and abdominal pain and thirst may be severe, but dryness and coldness not marked.

Stools. May be very frequent, up to 15 or 20 daily, but not pure blood.

Skin moist

Abdomen. Rarely retracted. Rigidity not uncommon, and contraction of muscles occurs on palpation at sites of tenderness. Sigmoid often palpable, contracted in spasm.

Tongue. Moist fur, or may be clean

Pulse rapid but not running.

Temperature variable.

Vomiting very rare.

Subsequent Progress.—Acute stage, four to five days. Rapid improvement in succeeding five days. Progress subsequently varies: may continue to improve rapidly, or drift into subacute and chronic types.

Mortality low.

NOTES ON SYMPTOMS.—

TEMPERATURE.—Not of great assistance. (*a*) *Severe forms*: commonly high at onset, but usually subnormal when condition has developed. (*b*) *Moderate forms*: temperature is some measure of severity. High temperature is a sign of severe infection, especially when persistent. Fall of temperature is a sign of improvement. Milder cases have slight pyrexia.

VOMITING.—Occasional vomiting at onset is common and of little importance. Persistence or onset of vomiting later is serious symptom.

SWEATING.—A sweating patient is rarely in immediate danger.

MILD FORMS.—Symptoms of any degree of mildness may result from infections with dysentery bacilli; condition often indistinguishable clinically from a simple transient diarrhœa.

Complications and Sequelæ.—

COLITIS.—Constipation, or alternating periods of diarrhœa and constipation, very common sequel. *Chronic colitis* may be a permanent sequel. Appendicitis is rare.

ARTHRITIS.—Onset usually during convalescence. Large joints, especially knees, affected. May occur in mild cases. Considerable effusion: fluid contains polynuclear leucocytes. *Complete recovery is invariable and suppuration never occurs*, but duration may be months. Heart unaffected.

IRITIS AND IRIDOCYCLITIS.—Especially with arthritis.

BOILS.—Occasionally troublesome.

HÆMORRHOIDS.—Occurrence common. Not uncommon cause of much blood in stools during convalescence.

PERITONITIS.—Perforation occurs rarely. In later stages and after severe attacks only. Peritonitis may be general or localized by adhesions. Perforation often multiple, and death-rate very high.

CICATRICIAL CONTRACTIONS. May cause intestinal obstruction. Rare.

TACHYCARDIA and various forms of disordered action of the heart develop occasionally.

PULSE-RATE IN CONVALESCENCE.—Bradycardia, 40 to 60, is not uncommon in 2nd to 4th weeks, especially in milder forms. Pulse-rate of 60 to 70 usual after more severe infections. About the 4th week, pulse-rate often increases to 100 or more rapid, as the patient gets up.

Convalescence.—After severe attacks, convalescence is very slow: many months. With moderate attacks, chills and dietetic errors rapidly cause intestinal disturbances. Dyspepsia and gastric discomfort common. Constipation frequent.

Diagnosis.—Diarrhœa of any form, mildness, or severity, may result

Bacillary Dysentery—Diagnosis, *continued.*

from infections with dysentery bacilli; but in epidemics characteristic cases will occur. The ultimate diagnosis depends on specific methods.

DIAGNOSIS FROM NON-DYSENTERIC CONDITIONS.—

1. ENTERIC.—Onset rarely acute. Mucus in stools unusual. Agglutination reactions and bacteriology.
2. FOOD-POISONING.—Characterized by simultaneous affection of many individuals. Condition is mainly ileitis or enteritis, and blood is unusual after initial severe motions, and mucus not prominent.
3. ACUTE ULCERATIVE COLITIS.—Now generally accepted that all forms are of bacillary origin. Indistinguishable clinically and pathologically from dysentery. Morgan's No. 1 bacillus not infrequent: exact relationship is in dispute.
4. MALARIA —'Malarial dysentery' is usually but not invariably true dysentery.

DIAGNOSIS FROM AMŒBIC DYSENTERY.—

	1. BACILLARY.	2. AMŒBIC.
Onset	Acute.	Often more gradual initial diarrhœa not uncommon.
Progress	Most severe at onset.	Irregular. Tends to be chronic.
Stools.—Often indistinguishable, but characteristically.	Single mass of glairy mucus, untinged by blood. Pus cells and blood present. Motions when formed are coated with mucus.	Mucus, blood, and fæcal matter more intimately mixed. Small masses of blood-tinged mucus. Motions when formed are mixed with mucus.
Complications ..	.. .. ..	Hepatic abscess.
Morbid Anatomy ..	Sigmoid most affected. Ileum often hyperæmic. Ulceration superficial. Mucous membrane thickened.	Cæcum and ascending colon mainly. Ileum rarely affected. Ulcers with undermined edges.

SPECIAL METHODS OF DIAGNOSIS.—

1. EXAMINATION OF STOOLS.—Examine for bacilli and also for amœbæ and amœbic cysts.
2. AGGLUTINATION.—Agglutinins usually appear early, by 2nd day, and usually maximum by 6th day. (1) *Shiga infections*: Agglutination often definite, 'positive' if in dilution 1–50 (microscopic test). (2) *Flexner infections*: Agglutination complicated by multiplicity of strains: also by tendency of normal sera to agglutinate these bacilli. Agglutinins are usually transient.

Prognosis.—*In severe forms*, as described above, mortality very high: 40 to 60 per cent. *In moderate forms*, mortality usually very low. The relative frequency of these two forms varies greatly with the causal bacillus. (*a*) *Shiga infections*: Severity is common, and convalescence prolonged even in milder forms; but simple diarrhœa may result from Shiga infections. *(*b*) *Flexner group*: In Europe, mortality from these infections during European War did not exceed 2 to 3 per cent.

Treatment.—

1. GENERAL TREATMENT.—The first essentials are *warmth and fluid*. If patient restless, wrap arms in cotton-wool and give extra shirt. *Water by mouth*: boiled water, ℥j every quarter to half hour. *Intravenous saline* with 5 per cent glucose if collapsed. For abdominal pain: turpentine stupe, hot-water bottle. Mouth wash: frequently.

2. DIET.—Small amounts: frequently (two to three hours): not hot, but without chill.

   IN SEVERE FORMS.—Fluids only. Whey, diluted milk, chicken broth. White-wine whey often well taken.

   IN OTHER FORMS.—Semi-fluids from onset or after one to two days. Milk, beef-tea or chicken-broth, custards, egg-flip, ice milk.

   PROGRESS.—As improvement occurs, amount of diet may be increased, but semi-fluid diet should be maintained until, for seven days, motions have not exceeded two daily and no visible blood or mucus is present. Proceed with boiled fish and then chicken.

3. MEDICINAL TREATMENT.—

   SALINE.—Sodium sulphate, ʒj, two-hourly for first day. Subsequently four-hourly and six-hourly for four to five days. Stools often improve rapidly and tenesmus is eased. The method aims at emptying the intestine. To be employed *only at onset of attack*, and is contra-indicated by numerous previous motions and in severe forms.

   When seen early, an initial dose of sodium sulphate ʒ[illegible] or castor oil may be given.

   ENEMATA.—*Starch and opium enema, especially for severe forms*: daily, very slowly through a catheter: retain as long as possible (press pad on anus). *Medicated enemata*: e.g., albargin (ʒss in a pint-and-a half of water), or tannic acid ʒij, not to be given in first few days, but valuable later, especially in chronic forms.

   DRUGS by the mouth have little effect. Mist. cretæ (B.P.) ʒj, two- or four-hourly. Bismuth salicylate gr. xx, t.d.s.

   MORPHIA.—Should only be given as last resort for extreme restlessness and insomnia. In general is *contra-indicated*.

   ALCOHOL.—Usually disliked and may cause vomiting.

   EMETINE.—Contra-indicated. Valueless in bacillary dysentery, and is an intestinal irritant.

4. SERUM TREATMENT.—To be given in all severe cases, but of little value except at onset. Inject 40 to 8[illegible] c.c. subcutaneously or in severe cases intravenously. A polyvalent serum must be used unless type of bacillus is known. *Serum reactions* are often severe.

CONVALESCENCE.—

   GASTRIC DISTURBANCES AND DIARRHŒA.—Modify diet, especially meat.

   CONSTIPATION.—Liquid paraffin ʒij, t.d.s., with simple enemata,

Bacillary Dysentery—Treatment, *continued*

e g, salt and water (ʒj to the pint) Avoid aperients, saline or vegetable

DIARRHŒA —Medicated enemata, e g, albargin A simple colonic wash, e g, salt and water, should be given in the morning, followed an hour later by the medicated enema

Prophylaxis.— General methods should be directed against modes of infections as in typhoid fever

'DYSENTERY CARRIERS' —Chronic carriers of bacillary dysentery are very rare Recognition is difficult owing to intermittency in excretion of bacilli.

INOCULATION —Reactions to dysentery vaccines have proved too severe for practice Efforts are being made to prepare a detoxicated vaccine.

II. AMŒBIC DYSENTERY.

Amœbic dysentery is caused by infection with the *Entamœba histolytica.*

The Amœba.—

ENTAMŒBA HISTOLYTICA - General characteristics

1 Size 15 to 50 μ diameter, commonly about 30 μ.
2 Clear refractile ectosarc with a granular vacuolated endosarc
3 Amœboid movements active Clear pseudopodia are thrown out and retracted
4. Often contains red cells
5. Nucleus indistinct and eccentric

Cysts.—

i Size 7 to 14 μ diameter Round
ii Nuclei 2 or 4 in number
iii Chromidial body present
iv. Cyst wall thin and indistinct

ENTAMŒBA COLI Size about same as, or rather larger than *histolytica.* Distinction often extremely difficult, depending on (i) absence of ectosarc, (ii) amœboid movements sluggish, (iii) red cells rare and never numerous, (iv) nucleus central and more distinct

Cysts —Distinction from *histolytica* depends upon —

i *Size*: diameter 15 to 20 μ sometimes 30 μ (Smaller cysts may occur; also *histolytica* are occasionally larger than 14 μ)
ii *Nuclei* 6 or 8, sometimes more Simplest and most reliable mode of distinction
iii. No chromidial body,
iv. Cyst wall more distinct.

ENTAMŒBA NANA (*Endolimax nana*).—A small (6 to 12 μ), non-pathogenic amœba The cysts are same size as *histolytica*, and contain 1, 2, or 4 nuclei, but are of oval shape.

PRESENCE OF ENTAMŒBA HISTOLYTICA IN STOOLS.—*Active forms* in acute stages only Stool must be examined immediately, as amœbæ rapidly disappear. Examine unstained or with a little weak neutral red, preferably on a warm stage. *Cysts*: Pick out portion of mucus: place on slide with Lugol's

iodine solution; this renders nuclei more distinct and also stains glycogen granules. Examine slide with a ⅛ lens and confirm with oil-immersion lens.

MODES OF SPREAD.—*Active forms* of amœbæ die very rapidly even in fæces. *Cysts* have long endurance in moisture, fæces, and water, but are rapidly killed by drying. Spread of disease is probably entirely by cysts, from presence in stools and frequency of 'carriers'. Flies are important cause of transmission by feeding on fæces and subsequently defæcating on food (Wenyon and O'Connor).

Morbid Anatomy.—*Cæcum and ascending colon* are usually most affected. The entire large intestine may be involved: less often sigmoid and rectum. The ileum escapes.

ESSENTIAL CHANGES.—(a) *Thickening of the wall*, mainly of the submucosa, by œdema and round cell infiltration; (b) *Ulceration*, occurring in thickened areas. The entire large intestine may be studded with ulcers, intervening areas of mucous membrane but little affected being practically always present. Amœbæ enter mucous membrane through crypts of Lieberkuhn, and mainly affect and spread in the submucosa.

INFILTRATION OF SUBMUCOSA.—Earliest stage due to œdema, multiplication of fixed cells, and round-cell infiltration. Polynuclear leucocytes are scanty at all stages. *Prominences* appear on gut, size of pea.

MUCOUS MEMBRANE over prominences necroses and sloughs, forming *ulcers* with irregular outline, ragged and characteristically *undermined edges*. Floor formed by any coat, even serous. In submucosa undermining of the mucous membrane is essential feature. Amœbæ are in the spreading edge.

HEALING results by formation of fibrous tissue. *Hence contractions and strictures occasionally result.*

All stages of ulceration and repair may be present simultaneously in the same specimen.

IN CHRONIC CASES.—Wall thick in some parts, in others thin, scarred, and pigmented. Cicatricial contractions and peritoneal adhesions may be present.

PERFORATION AND PERITONITIS may occur.

LYMPHATIC GLANDS.—Usually enlarged.

LIVER ABSCESS.—Occurs in 2 per cent of cases. Commonly single, in right lobe and on diaphragmatic surface. Occasionally two or more present. May occur with very slight attacks of dysentery. Early abscess: contents gray yellow. Larger abscess: necrotic walls, contents reddish mass of blood and liver tissue. Old abscess has dense, fibrous walls.

Contents are sterile (in absence of secondary infection), are not purulent, and consist of detritus. Amœbæ only present in recent abscess: in old abscesses only found in walls.

Rupture into lung common, leading to anchovy-sauce sputum.

Symptoms.—

INCUBATION PERIOD.—Symptoms are recorded within two days, but data are scanty.

Amœbic Dysentery—Symptoms, *continued.*

ONSET.—Previous diarrhœa is common and onset gradual. Acute onset occurs occasionally.

SYMPTOMS IN GENERAL resemble bacillary dysentery, but condition is characterized by greater irregularity and intermissions, by tendency to chronicity, and by occurrence of complications, also by frequency of 'carriers'. Pyrexia is frequently absent. Very severe forms and toxæmic symptoms are unusual.

PROGRESS.—Severe forms may resist treatment and be fatal in 7 to 10 days. Recovery from acute stage is more common, and initial mortality is low. Subsequent conditions:—

1. Long convalescence with alternating diarrhœa and constipation.
2. Chronic condition of diarrhœa and dysenteric stools: may be fatal after several months.
3. Complications: (i) Hepatic abscess, (ii) Perforation and peritonitis. Rarely hæmorrhage.
4. Chronic carrier of cysts.

MILD FORMS.—Common. May be slight diarrhœa without other symptoms. Such cases not uncommonly become carriers

Diagnosis, and Character of Stools.—*See* BACILLARY DYSENTERY.

SPECIAL METHODS.—Examination of stools for *Entamœba histolytica* and cysts.

Complications.—Important.

LIVER ABSCESS.—Occurs in 2 per cent. Symptoms of dysentery may be very mild or unrecognized. For symptoms, *see* ABSCESS OF THE LIVER.

PERFORATION AND PERITONITIS.—Usually in chronic stage of severe attacks. Mortality high owing to extensive lesions of intestine. *Hæmorrhage* rare, but may be fatal.

Other complications as in bacillary type, but no arthritis.

'Carriers'.—Cysts of *Entamœba histolytica* present in stools. May have had severe attack, especially with insufficient treatment with emetine. Often, attack of dysentery very slight or unnoticed.

Treatment.—General treatment, diet, etc., as in bacillary dysentery, but saline treatment with sodium sulphate is contra-indicated. Serum is valueless, but harmless.

EMETINE.—Essential in all forms of *Entamœba histolytica* infections. Rogers introduced the drug. The original method was by hypodermic injections of emetine hydrochloride. This is effective in checking initial symptoms, but subjects frequently become carriers, and on these this method has little value.

EMETINE-BISMUTH-IODIDE.—Recommended by Du Mez, 1915: brought into use by Dale. More effective remedy, especially in treatment of carriers. *Dosage*: three grains daily for twelve days, by the mouth, in gelatin capsules. If relapse occurs, give a second course for twenty-four days.

The drug is a gastric and intestinal irritant, causing nausea,

often vomiting, and less often diarrhœa Best administered in a single dose the last thing at night, and patient told to lie still

Improvement in acute cases is very rapid under emetine treatment, and mortality low Is valueless and contra indicated in bacillary dysentery

VARIOUS INTESTINAL INFECTIONS.

Lamblia (or *Giardia*) *intestinalis*—A flagellate protozoon, which inhabits the duodenum and jejunum Pyriform in shape, with a characteristic saucer shaped depression and four pairs of flagella length about 20 μ Encysted non flagellated form also occurs Relation to diarrhœa denied by some competent authorities often present in stools of healthy individuals Most frequently present (often in enormous numbers) in a diarrhœa of lienteric type with large yellowish stools Never causes dysenteric symptoms Bismuth salicylate procures temporary absence, but no drug causes their complete removal (Dobell and Low)

Trichomonas intestinalis *Tetramitus mesnili* — No evidence of pathogenicity

Balantidium coli or *Paramecium coli*—No proof of pathogenicity

CHAPTER XI

MALTA FEVER.

(*Mediterranean Fever Rock Fever. Undulant Fever.*)

An infective disease of long duration caused by the *Micrococcus melitensis*, and characterized by a series of pyrexial attacks, with constipation, muscular pains, arthritis anæmia, and enlarged spleen Infection conveyed by goat's milk

Geographical Distribution.—Shores of the Mediterranean, with foci in tropics and goat rearing districts.

Bacteriology. *Micrococcus melitensis* was discovered by Bruce in 1886, obtained post mortem from blood and spleen, and its relation to the disease stablished by inoculation experiments

MORPHOLOGY.—Very minute coccus, occurs singly, in pairs, or (in cultures) in short chains Non-motile. Stains with ordinary stains *Gram negative.*

CULTURAL CHARACTERISTICS—Grows on ordinary media, but colonies not visible before 3rd day Does not ferment dextrose Renders milk and other media alkaline.

OCCURRENCE IN BODY.—Numerous in *spleen*. Present in blood during attack. Excreted in *urine*, after 15th day in 10 per cent of cases, usually for many weeks and may be for many months • At autopsy · isolated from spleen

AGGLUTINATION REACTION.—Present throughout disease, commencing about 5th day. Serum, during disease, agglutinates

Malta Fever—Bacteriology, *continued.*

micrococcus in high dilutions, frequently 1–500. Agglutination, in lower dilutions of serum, may persist for long periods, 2 years or more. Careful controls are essential, as the organism may agglutinate spontaneously: also normal serum contains some agglutinins.

Mode of Infection in Milk.—Mediterranean Fever Commission, 1904, discovered that the micrococcus is present in the milk of 10 per cent of the goats in Malta: directed to this by discovery that the blood of 50 per cent of the goats agglutinated the organism. Goat's milk was drunk extensively: since its exclusion, the disease has disappeared from the troops in Malta. Probably this is the sole general method of infection, no spread being traced to the excretion of micrococci in human urine. Laboratory infection occurs with great ease and frequency.

OCCURRENCE IN GOATS.—Mode of infection uncertain. Infected goats may appear healthy, but after some months become thin, and milk poor.

Monkeys and other animals are readily infected.

One attack apparently confers immunity.

Morbid Anatomy.—*Spleen* weighs about 1 lb., soft and congested. No other characteristic changes. Alimentary tract nil.

Symptoms.—The condition is a septicæmia, characterized by irregular undulations of temperature.

INCUBATION PERIOD.—Usually about fifteen days, but limits uncertain, at least six to twenty days.

EARLY SYMPTOMS.—Malaise, often muscular pains and gastric disturbances. These may persist throughout.

CHARACTERISTIC ATTACK.—*Period of fever* with symptoms lasting one to three weeks. *Period of defervescence* follows: may be slight pyrexia or normal temperature and convalescence from ten to twelve days. *Relapse* occurs for shorter period. *Longer apyrexial period,* which may be again followed by yet milder relapse. Number of undulations variable, often three in mild case: may be numerous. *Duration*: very variable and course erratic; often three to six months, but may be more prolonged, two years.

MODERATE ATTACK OR FIRST UNDULATION.—(1) *Pyrexia,* 102° to 104° or 105°, typically step-like rise and fall, but may be markedly irregular or even intermittent. (2) *Gastric disturbance.* Constipation obstinate. Nausea and vomiting not infrequent. Diarrhœa occasionally. (3) *Profuse sweats.* (4) *Muscular pains.* (5) *Headache,* restlessness. (6) *Spleen* enlarged and tender.

SEVERER SYMPTOMS.—Occur more frequently during relapses. (*a*) *Headache* severe. (*b*) *Arthritis*: may be large effusion. Tends to be transient, but reappears in other joints. No redness. Pains may be agonizing. (*c*) Neuralgia, pains and sciatica. (*d*) *Fibrositis.* Especially round ankle-joint. (*e*) *Anæmia*: progressive. (*f*) Insomnia.

OTHER SYMPTOMS —Rashes rare: erythema, rarely purpura Bronchitis and lung affections occur in late stage Orchitis and epididymitis rare but painful

Progress.—When relapses are numerous great debility and mental depression develop, with anæmia and tachycardia

Varieties. – (1) MILD FORM Evening pyrexia and slight malaise severe symptoms may develop (2) MALIGNANT FORM Fatal in one to two weeks Very rare

Diagnosis. Often difficult clinically Lymphocytosis common
BLOOD CULTURES Not infrequently negative
SPLEEN CULTURES Micrococcus isolated
AGGLUTINATION REACTION —Specific.

Prognosis. Mortality low 2 per cent

Treatment. *General Treatment* as in typhoid fever No specific treatment exists Quinine and salicylates of no effect Local applications to joints Change of climate after acute stage *Vaccine Treatment* is under trial

CHAPTER XII.

CHOLERA ASIATICA.

An acute infective disease due to presence of the cholera brio in alimentary tract, and characterized by purging, muscular cramps, and rapid collapse Infection is usually water borne

Etiology. –
CLIMATE Endemic and epidemic in tropics Prevalence greatest in India In temperate zones occurs as epidemics, but never endemic
SEASON Favoured by hot weather in temperate zones, especially in early autumn
AGE All ages affected One attack does not confer immunity.

Bacteriology. Organism discovered by Koch in 1883 in outbreak in Egypt, known as *cholera vibrio, cholera spirillum,* or *comma bacillus*
MORPHOLOGY —Small, mottle, curved rods, about 2 μ long In cultures mostly singly, but two may join together like an S. A single terminal flagellum is usually present, but in some varieties may be two, as in Massonah strain. Correctly it is a spirillum, and in liquid media growth tends to spirillar forms The short forms are called vibrios. In old cultures numerous involution forms are seen, many are circular but are not spores. *Gram-negative,* but stains with ordinary stains, preferably weak carbolfuchsin, one part to four of water.

Cholera Asiatica—Bacteriology, *continued.*

CULTURAL CHARACTERISTICS. – Grows on all ordinary media. Characteristic are : –

1. GELATIN STAB.—On 5th day air-bubble on surface, with funnel of liquefaction below.
2. GELATIN PLATES. - Colonies have granular surface with irregular outline like fragments of broken glass. Later, medium liquefies, with appearance of concentric rings.
3. CHOLERA RED REACTION.—Growth in broth forms both indol and nitrite. Addition of pure sulphuric acid gives a pink colour from nitroso-indol. The culture must be 8 days old. Reaction increases up to 2 or 3 days. Not all broth preparations give reaction. Sulphuric acid used must be free from nitrites.

On broth growth forms a surface pellicle. In milk grows well without apparent change in medium.

DISTRIBUTION IN THE BODY.—Essentially in the intestines. Vibrios do not penetrate deep in mucosa. Occasionally in gall-bladder ; very rarely in other organs (and even recorded in blood). Numerous in motions, especially in rice-water stools, which may contain almost pure culture. Symptoms probably due to absorption of toxins. In preparations from stools, organisms tend to lie with long axes parallel, 'like fish in a stream'.

RESISTANCE.—Life in ordinary drinking water very variable, and depends partly on the temperature and amount of organic matter present. Varies from a few days to three weeks. Can multiply in water. *Drying* kills in a few minutes. Can live several weeks on moist linen. In stools, rapidly overgrown by bacilli.

SPECIFICITY OF KOCH'S CHOLERA VIBRIO. - Now generally accepted as cause of cholera. Fulfils Koch's postulates. is constantly present in cholera, can be isolated and grown pure in subculture, pure cultures will reproduce the disease.

AGGLUTININS, ANTISERA.—*Agglutinins* appear in blood eight to ten days after onset, and reach maximum in two to four weeks, agglutinating cholera vibrio in high dilution. Consequently of no value *for immediate diagnosis*, but agglutination is usually positive in cholera carriers.

Antisera have been prepared. No obvious value in treatment. Valuable in identification of cholera cultures.

ANTI-CHOLERA INOCULATION.—Protection high, but should be repeated every 5 months. *No reaction occurs.* Incidence among inoculated is low : case-mortality is less influenced. Inject 12,000 to 15,000 million bacilli : two inoculations at intervals of 7 to 10 days.

BACTERIOLOGICAL DIAGNOSIS.—

1. Prepare a film from the stools and stain with weak carbol-fuchsin. Organisms may be present in large numbers.
2. Inoculate broth with loopful of stools. Incubate for two hours, and subculture into media as described under cultural characteristics.

3. Agglutination of cultures with specific cholera antiserum. Pfeiffer's reaction: a suspension of cholera vibrios mixed with anti-cholera serum is injected intraperitoneally into guinea-pigs and the lytic action examined.

Mode of Infection. Is essentially water-borne, and all large epidemics are spread thus. Infection may be due to (i) *Water.* Drinking water undoubtedly most common factor. Also by vegetables, etc., washed in infected water. (ii) *Cholera carriers.* Virulent vibrios may be present in the motions of clinically healthy persons. *Food* may be thus infected by cooks, or water supply affected. Usually transient, 1 to 2 weeks, rarely 2 months. Vibrios in fæces of patients rarely longer than 3 weeks, frequently only a few days. (iii) *Flies* may carry infection to food. *Direct contagion slight.* Doctors and nurses rarely affected. Is not air-borne.

Symptoms.—

INCUBATION PERIOD. A few hours to a few days. May be slight diarrhœa and malaise.

Clinical course usually described in three stages: (1) Stage of evacuation; (2) Stage of collapse (algid stage); (3) Stage of reaction.

STAGE OF EVACUATION.—*Onset abrupt.* (i) *Severe purging,* followed rapidly by (ii) *Vomiting*—often becomes incessant. (iii) *Muscular cramps,* especially in legs; may be agonizing. (iv) *Progressive exhaustion.* (v) *Thirst* becomes extreme. *Stools* at first yellow, rapidly become white, so-called rice-water stools. When frequent, usually odourless. *Tenesmus* usually absent. *Temperature* generally subnormal. *Pulse* feeble. Exhaustion and collapse increase. Consciousness retained. Recovery may now commence, or more advanced collapse follow.

STAGE OF COLLAPSE. ALGID STAGE. *Collapse extreme,* face pinched, eyes sunken, skin wrinkled, restlessness, cyanosis, clammy perspiration, semi-consciousness or coma. Involuntary passage of watery motions; may be anuria. *Temperature* subnormal; may be high in rectum. *Pulse* rapid, may be impalpable. *Duration,* from two or three to twenty-four hours. *Mortality* very high. The collapse is due to withdrawal of fluid from the blood, resulting in concentration; the specific gravity of the blood rises to 1060 and may reach 1072 or 1078 (normal 1058). Blood thick. Pressure low, 70 mm. or under.

STAGE OF REACTION. In favourable cases with or without algid stage. Rapid improvement. Consciousness returns. Skin becomes warm. Bile appears in motion. Stools become less frequent. Usually some fever. Erythema common.

CHOLERA TYPHOID.—Stage of reaction may be incomplete, and a typhoidal condition develop, usually with anuria. Common towards end of first week in severe cases.

CONVALESCENCE.—Usually rapid. Complications arising may be: *Recrudescences,* frequent and often fatal; *Erythema* and numerous forms of skin eruptions, may be hæmorrhagic.

Cholera Asiatica—Symptoms, *continued.*

SEQUELÆ.—Unusual, recovery generally complete: (1) *Nephritis.* (2) *Cramps in muscles.* (3) Diphtheritic inflammations of mucous membranes, of intestine, fauces, and genitals. (4) Various results of weakness: (*a*) Psychical, e.g., insomnia; (*b*) Tendency to boils, pneumonia, etc.

TYPES.—All grades of severity occur. In mild types or *cholerine*, collapse slight but vibrios present in dejecta. In most severe form, *cholera sicca*, purging absent and death very rapid.

Diagnosis.—During epidemics diagnosis simple. In sporadic cases confusion may arise with arsenic and food-poisoning, and certain acute bacillary affections.

Cholera nostras.—In severe epidemics of summer diarrhœa, symptoms may resemble cholera. Difficulty arises especially in children, in whom also cholera is often atypical. Stokers' or firemen's cramp, caused by drinking cold water when heated, has similar symptoms. Diagnosis in these conditions by bacteriological methods (q.v.), microscopic examination of stools usually being sufficient.

Prognosis.—Unfavourable with very rapid onset, low temperature, and especially with high specific gravity of blood, 1065 or over. Mortality formerly about 70 per cent, but greatly diminished by Rogers' method of saline infusions.

Prophylaxis.—*Preventive methods* in checking epidemics: (1) Isolation of patients and disinfection of excreta; (2) Search for 'cholera carriers'. For individuals, there are three important considerations: (i) Keep the general health good: especially attend to diet, avoiding over-ripe fruit. Treat any diarrhœa promptly. Avoid alcohol, especially on empty stomach. (ii) Boil all water and milk, and protect all food from flies. (iii) Inoculation with anti-cholera vaccine.

Treatment.—

GENERAL TREATMENT. Rest in bed and warmth. Give water by the mouth. In early cases a preliminary dose of castor oil ʒj may be given. Powerful drugs to check diarrhœa must not be given, and morphia injections are contra-indicated.

DIET.—Food is of no value; give brandy, hot coffee, or ice alone by the mouth. Diet carefully in the stage of reaction to avoid relapse.

FOR THE CRAMPS.—Gentle massage and hot fomentations. When very severe, a whiff of chloroform.

CARDIAC WEAKNESS.—Injections of camphor eight-hourly (gr. ij in ♏x of sterile olive oil).

FOR ANURIA.—Fomentations to the kidneys. Normal saline per rectum frequently. Injections of pituitrin. For uræmia, sodium bicarbonate intravenously.

ROGERS' METHOD.—(1) *Hyperionic intravenous saline injections* are of highest value. Indicated in severe forms and when specific gravity of blood exceeds 1063. The formula is: sodium chloride gr. cxx; potassium chloride, gr. vj; calcium chloride, gr. iv; water

one pint. Give three or more pints at temperature 98° or lower intravenously at rate of 4 oz. a minute. This may be repeated several times at intervals of a few hours. (2) *Potassium permanganate*, 2-gr. keratin-coated pills, every 15 minutes for 4 hours, then half-hourly until motions green. Calcium permanganate, gr. ½ to the pint, in large draughts.

Powdered kaolin and charcoal have had some good results.

Other Species of Vibrios.—Numerous species of vibrios have been isolated in varying circumstances.

PARACHOLERA. Strains have been isolated from stools in diarrhœa or mild cases of cholera. Distinguished from Koch's vibrio by agglutination with antisera. Mortality is very low and epidemics do not occur.

Certain strains isolated from patients with dysenteric symptoms have agglutinated with antisera to Koch's vibrio, e.g., *El Tor vibrio*. Identity or otherwise not yet certain.

METCHNIKOFF'S SPIRILLUM.—Isolated from epidemic in fowls. Pathogenic to pigeons and animals.

FINKLER-PRIOR'S SPIRILLUM.—Isolated from acute diarrhœa in children (cholera nostras). Pathogenicity not proved.

CHAPTER XIII.

PLAGUE.

A specific infective disease caused by *B. pestis* and conveyed by rat-fleas, and occurring in three clinical forms, bubonic, pneumonic, and septicæmic; of which the two former occur in vast epidemics.

Etiology.—Present cycle commenced in Hong Kong in 1894.

MODE OF SPREAD.—The principal factors are briefly as follows: (1) Disease primarily affects rats, and in these is always septicæmic. (2) Rat-fleas suck blood containing bacilli. (3) Rat-fleas attack man and inoculate when biting. (4) Spread among rats is due to rat-fleas, cannibalism, and possibly human fæces and infected food. (5) From rat to man, infection is solely by fleas. Infection is very rare directly from man to man. Spread of epidemic is practically entirely due to spread in rats and thence to each human being individually. Drinking water apparently of no influence. (6) Epidemic is always preceded by epizootic in rats or, rarely, other ground animals, e.g., ground-squirrel in Californian epidemic. Outbreak in animals in a district precedes human cases by about two weeks.

Rat-fleas are *Pulex cheopis*, most frequent in tropics, and *Ceratophyllus fasciatus*, most frequent in temperate regions: the latter bites man less readily. Infection is due to regurgitation of infected blood from the stomach while biting (C. J. Martin).

Plague—Etiology, *continued.*

PNEUMONIC PLAGUE forms an exception to some of above statements Spreads directly from man to man. Bacilli present in sputum in large numbers. Spread very rapid, but life of bacilli outside body very short, hence no epizootic of rats occurs, and epidemic may be rapidly extinguished.

DISTRIBUTION.—Mainly a disease of tropics, but few countries have entirely escaped since present cycle commenced in 1894. In England, several small outbreaks in Suffolk. Rats in seaports in several countries are being systematically examined and plague-infected animals occasionally discovered. *Frequency* greatest in cool weather in the tropics, and in hot weather in temperate regions.

Bacteriology. *B pestis* isolated by Kitasato and by Yersin in 1894

MORPHOLOGY.—Short fat bacillus with rounded ends and marked 'polar staining'. Non-motile and non sporing Stains with usual stains, but *Gram-negative* Numerous involution forms occur in cultures, especially on Hankin's 'salt agar', agar containing NaCl. In tissues, mainly single: in liquid media may form chains

CULTURAL CHARACTERISTICS Grows on agar and ordinary media. Most characteristic is Haffkine's 'stalactite growth' in butter fat broth Killed readily by heat and antiseptics Old cultures lose virulence, but regain it on subculture

METHODS OF ISOLATION.—(*a*) Bubonic plague: Puncture bubo with hypodermic needle, make and stain smears, and inoculate media. (*b*) Pneumonic plague. Smears from sputum, inoculate media. (*c*) Septicæmic type. Culture from blood, sometimes seen in blood films. Post mortem, bacilli present in every organ

AGGLUTINATION REACTION—Agglutinins appear about end of first week, but titre is not very high, and agglutinins often absent in severe and very mild forms Reaction also complicated by frequent spontaneous agglutination of cultures. Results to be interpreted with care and only by experienced workers.

SUSCEPTIBILITY OF ANIMALS.—Guinea-pigs, mice, rats, rabbits, and most animals are susceptible. *Subcutaneous inoculation* results in: (1) Œdematous swelling at site of inoculation; (2) Nearest lymphatic glands enlarge, hæmorrhages present; (3) Septicæmia: bacilli present in blood. Death usually in two to four days. Bacilli in most tissues, especially spleen.

In monkeys: may be no local swelling at site of inoculation.

Morbid Anatomy.—

BUBONIC TYPE.—*Enlargement of lymphatic glands*, usually commencing in one group, most commonly axillary or inguinal, forming the 'primary bubo'. Other groups subsequently enlarge, forming 'secondary buboes,' but to less extent. *Bubo*: Inflammation of glands, with extensive periglandular œdema; on section, hæmorrhages present, in early stages, masses of bacilli; later, advanced necrosis of cells, bacilli often few or absent.

Suppuration not uncommon, but does not occur until second week, and hence never in the rapidly fatal cases. *Hæmorrhages* and focal necroses common in other organs, and cloudy swelling.

PNEUMONIC TYPE.—Patchy bronchopneumonia and areas of red hepatization. Bronchial glands enlarged.

SEPTICÆMIC TYPE.—General appearances of septicæmia with hæmorrhages.

SPLEEN.—Commonly enlarged.

SKIN.—Hæmorrhages may be either *petechial* or *diffuse and extensive*. Over a bubo, the skin may be discoloured by hyperæmia.

Symptoms.

INCUBATION PERIOD.—2 to 5 or possibly 10 days. Usually no symptoms. May be malaise. *B. pestis* has been found in blood.

CLINICAL TYPES.—(1) Bubonic; (2) Pneumonic; (3) Septicæmic. Bubonic is the commonest epidemic type.

1. BUBONIC PLAGUE. *Sudden onset*: chill, headache, backache, restlessness, rapid pulse and respiration, high fever. Symptoms often fully developed in a few hours. Great prostration occurs rapidly, and often a typhoidal condition within one to two days. *Bubo*: usually in one to two days from onset. Femoral glands most common, next axillary. Cervical not uncommon in children. Swelling size of egg or larger. Very tender. Œdema may be extensive. Fever may fall slightly on appearance of bubo. Secondary buboes form later. Spleen usually palpable.

   Symptoms usually *progress*: extreme prostration and cardiac weakness, tongue brown, sordes, vomiting common, and delirium. Death in two to seven days: usually three or four. Mortality at least 75 per cent.

   In *favourable cases*, symptoms improve after bubo appears. In second week suppuration or resolution occurs. Prognosis improves after fifth day.

   In certain epidemics, *petechiæ and hæmorrhages* common ('plague spots'). Hæmorrhages from mucous membranes in severe cases.

   In children, convulsions at onset often so severe as to mask diagnosis.

   Blood: polynuclear leucocytosis. Bacilli often numerous before death.

   Temperature: High at onset, 103° to 104°. Subsequent course variable: not uncommonly falls after three to four days, and rises rapidly again in one to two days.

   During convalescence, a tragic fatal cardiac failure is common. Prolonged tendency to boils.

2. PNEUMONIC PLAGUE.—*Sudden onset*: Rigors, pain, cough, fever, and extreme prostration. Rapid pulse and respiration. Cyanosis. Sputum watery and bloody. Patchy consolidation in both lungs. Spleen palpable. Invariably fatal in one to four days. Numerous bacilli in sputum.

Plague—Symptoms, *continued.*

3. SEPTICÆMIC TYPE—All forms of plague become septicæmic, but this type specially includes cases without bubo or local signs. General symptoms severe and death invariable, frequently in one day. Hæmorrhages common. Does not occur as distinctive epidemic.

PESTIS MINOR. Slight cases occur, especially towards end or beginning of epidemic and in inoculated persons. Bubo may form. Death from cardiac failure may occur.

Diagnosis. During epidemic easy. When suspected, bacteriological proof simple. Early cases in epidemic easily overlooked. Suspect *outbreaks of rapidly fatal pneumonia, especially with several cases in one household*; also buboes from tropics and seaports. In tropics buboes occur from filariasis and also from unknown causes; also from syphilis and suppuration.

Treatment.—Careful nursing. To bubo, ice or fomentations; incise when fluctuating; injections into glands harmful. For mental symptoms, bromides. *Yersin's serum* in large doses possesses some value. During convalescence, *avoid slightest cardiac strain.*

PROPHYLAXIS. *Vaccine treatment.* *Haffkine's prophylactic* vaccine is of great value, and gives considerable immunity for a few months (three to six). All contacts should be inoculated. In small outbreaks, all contacts must be isolated, bedding and clothing burnt, and houses rendered airtight and disinfected with burning sulphur.

In large epidemics, a wide organization is necessary. The destruction of rats and examination of their bodies for bacilli, cleanliness of houses, and protection of uninvolved areas by quarantine are initial measures.

Quarantine Period—Ten days.

CHAPTER XIV.

TETANUS.

(Lockjaw.)

An infective disease caused by the toxins of *B. tetani*, and characterized by spasms of the voluntary muscles, commencing usually in the jaw and neck, and extending to the rest of the body.

Etiology.—Occurs as a sequel to wounds and abrasions throughout the world wherever soil is cultivated and manured. Under equal conditions, more common and more severe in the tropics. *Warfare* in cultivated regions is always accompanied by tetanus. In the European War, it was prominent among all armies until greatly controlled by prophylactic injections.

ACCIDENTAL INFECTIONS have occurred repeatedly from injections of gelatin, guaranteed freedom from tetanus spores being seemingly impossible; also, very rarely, from catgut.

'IDIOPATHIC' or 'RHEUMATIC' TETANUS.—Formerly applied to tetanus occurring without a visible wound. Now recognized that tetanus bacillus or spores may enter through apparently unbroken skin (as in 'trench feet'), and also may remain for prolonged periods before symptoms occur.

Bacteriology.—*B. tetani* discovered by Nicolaier, 1885, and isolated by Kitasato, 1889, in pure culture anaerobically.

MORPHOLOGY.—Slender bacillus. Forms a terminal spore wider than bacillus, thus producing characteristic 'drum-stick' appearance. Stains with ordinary stains. Gram-positive. Weak methylene-blue, followed by carbol-fuchsin, stains bacillus blue and spore as a red ring. Slightly motile. Numerous flagella: need special stain. When spores present, bacilli may be recognized in pus. Some of the 'gas gangrene' bacilli are closely similar, but shorter and thicker, and spores rarely quite terminal.

CULTURAL CHARACTERISTICS.—*Strict anaerobe.* Isolation very difficult owing to simultaneous presence of other spore-bearing anaerobes. Methods mainly depend on resistance of spore to heat and subsequent growth anaerobically on numerous subcultures, trusting that one may be in pure culture.

Spores extremely resistant to heat or antiseptics; resist boiling for five minutes. Virulent for many years in dried cultures.

OCCURRENCE OF BACILLI. Constantly present in intestines of horses and in their excreta. Consequently present in all heavily cultivated soil, especially a few inches below surface.

DISTRIBUTION OF BACILLI IN TISSUES.—Bacilli are present only at site of inoculation or in wound: practically never, if ever, present in organs or blood. Action is therefore due to toxin produced. The original wound is usually septic, the destruction of tissue by other organisms producing a suitable anaerobic medium for tetanus bacilli.

TETANUS TOXIN.—Injection of a filtered culture, i.e., pure tetanus toxin, produces all symptoms of tetanus. Toxin is highly potent. Ehrlich demonstrated presence of two types of poison: (1) Tetanospasmin, producing spasms; (2) Tetanolysin, hæmolytic to red cells.

MODE OF ACTION.—

An *incubation period* is always present between injection and onset of symptoms, even with enormous doses. The period varies with dose and mode of injection, but, for similar methods, varies mainly with size of animals, e.g., guinea-pigs few hours, monkeys about four days, horses about five days.

Meyer and Ransom's Experiments. (1) Tetanus follows injection into a motor (or mixed) nerve, but no symptoms result if nerve be divided proximal to site of injection previously or shortly afterwards (one hour). (2) No symptoms follow injection into a pure sensory nerve, e.g., infra-orbital. (3) If toxin be injected into a sensory nerve root, extreme hyperæsthesia with agonizing pain occurs in the corresponding area, but without spasms (tetanus dolorosus); hence the toxin can act on sensory nerve tissue.

Tetanus—Bacteriology, *continued.*

Conclusions.—The toxin is absorbed by muscle end-plates, and travels by motor nerves to the central nervous system, where it combines with nerve tissues and symptoms commence. There is no transmission by sensory nerves. An incubation period is unavoidable during passage of toxin along nerves. After reaching the spinal cord, toxin ascends in it

Toxin in the Blood.—A certain amount circulates in the blood and directly reaches the medulla and pons, producing generalized tetanus. Amount and effect of this varies in different mammals; apparently none in guinea-pigs.

SUSCEPTIBILITY OF ANIMALS. Nearly all animals are susceptible, degree varying greatly. Hen needs enormous dose. Alligator is completely immune, toxin probably unable to combine with nerve tissue. Horse is very highly susceptible. Monkeys, mice, guinea-pigs also highly, but less than horse. Symptoms follow injection of bacilli, spores, or toxins, but not by feeding. In small animals, spasms commence in muscles nearest site of inoculation. In frogs, no symptoms occur after inoculation until warmed in an incubator to 37° C.

Mice: For testing discharges from wounds, introduce portion of pus into root of tail

ANTITETANIC SERUM.—

PREPARATION AND IMMUNIZATION OF ANIMALS.—Animals can be immunized by injections of toxin, preferably by a toxin weakened by heat or keeping, by treatment with iodine, or by simultaneous injection of iodine trichloride. The serum of the animal has antitetanic properties, injections protecting against a subsequent lethal injection of toxin, or, in certain circumstances, against a previous injection, depending on mode of injection of serum and interval elapsing (*see also below*).

STANDARDIZATION OF SERUM.—Now standardized as 'U.S.A. units', corresponding to Ehrlich's unit for diphtheria serum, viz., one unit of serum protects against 100 'minimal lethal doses' (M.L.D.) of toxin, tested by mixing toxin and serum, injecting subcutaneously into standard guinea-pigs (250 grm.), and animal being still alive after four days. Serum now produced in two strengths: (1) 150 units in 1-c.c., or 1500 units in 10-c.c. phial; (2) 800 units in 1 c.c. Potency is maintained for long periods

ACTION OF SERUM.—Meyer and Ransom's experiments: (1) Antitoxin injected into a nerve prevents the passage of a distal injection of toxin into the nerve; (2) Antitoxin injected intravenously has no effect on a nerve injection of toxin; (3) An immunized animal can be killed by a nerve injection of toxin.

Conclusion: Antitoxin injected into the circulation only neutralizes circulating toxin.

FOR CURATIVE PURPOSES.—*In clinical tetanus*, serum is disappointing. Mainly due to absence of symptoms until toxin has reached central nervous system—no local lesion occurring comparable to sore throat of diphtheria—and to inefficiency of serum when this has taken place.

TYPES OF TETANUS BACILLI.—Several types of bacilli have recently been separated by serological tests. It is considered that an antiserum is only effective against its own strain, and hence that antisera for clinical use should be 'polyvalent' against all types.

Morbid Anatomy.—No characteristic changes.

Tetanus among British Troops during the War.—

Prevalence of tetanus was high in the early months in France, due to the extensive lacerated wounds and contamination with cultivated soil.

COMPARISON OF SERIES OF CASES.—Comparative examination of different forms of treatment is rendered very difficult by number of factors involved. The following points may be indicated:—

1. VARIABILITY OF INDIVIDUAL CASES.—Variations occur from (i) Size of wounds; (ii) Degree of sepsis and general condition of patient; (iii) Length of incubation period; (iv) Day of commencement of treatment; (v) Amount of toxin present—cannot be estimated; (vi) Auxiliary treatment—rest, sedatives, and diet is of great importance.
2. DURATION OF DISEASE.—Milder cases with longer duration obviously tended to receive larger doses of antiserum, complicating statistics of dosage.
3. COMPARISON OF CASES TREATED IN FRANCE AND ENGLAND.—The wounded rapidly transferred to England tended to be less severe and with longer incubation period.

War Office Committee under Bruce studied the questions of tetanus arising in the War. Findings of special importance are:—

A. PROPHYLACTIC INOCULATIONS OF SERUM.—Recommendations, briefly: (i) All wounded to receive an injection of 500 to 1500 units at earliest moment. (ii) Subsequently four injections at intervals of seven days: based on rapid fall of immunity after seven to ten days. (iii) 'Trench feet' to be included, even in absence of abrasion of skin. (iv) Injection two days *previous to any operation*, even if wound closed and healed, owing to lingering of tetanus spores.

RESULTS.—(1) *Deaths from tetanus greatly reduced.* After trench feet, practically abolished. (2) Incubation period prolonged. Previous to prophylactic injections, periods exceeding twenty-two days were rare, about 5 to 7 per cent in early months of European War. Proportion subsequently rose to 40 to 60 per cent for wounded transferred to England, but remained much lower for severely wounded retained in France. (3) Localized tetanus not infrequent; death-rate in this very low.

B. CURATIVE TREATMENT WITH SERUM.—Principal problems: (a) Mode of injection; (b) Dosage. Facts universally admitted: (a) Treatment should commence immediately on diagnosis; (b) Doses should be large.

Tetanus—Serum Therapy in War, *continued.*

a. MODES OF INJECTION are: (1) Intramuscular; (2) Subcutaneous; (3) Intravenous; (4) Intrathecal. Comparative value in practice much disputed.

SHERRINGTON'S EXPERIMENTS ON MONKEYS. A series of monkeys was inoculated with similar doses of toxin, and two to three days later, after commencement of symptoms, injected with antiserum. *Recoveries* by various methods: (1) Subcutaneous, 8 per cent; (2) Intramuscular, 12 per cent; (3) Intravenous, 28 per cent; (4) Intrathecal, 56 per cent.

NOTES ON THE METHODS.—(1) *Subcutaneous and intramuscular.* Absorption of serum slow: maximum concentration in blood not until forty-eight hours later. Advantage: simplicity, possibility of injection near wound. Clinical statistics definitely favour these methods, especially intramuscular. (2) *Intravenous.* Absorption rapid, also elimination rapid. Large doses possible. Anaphylaxis may occur and be fatal (not in other methods): partly prevented by general anæsthetic. Statistics inconclusive. (3) *Intrathecal.* Theoretically, rapidly immunizes the tissues of the central nervous system. General anæsthetic necessary. Amount of serum injected is very limited. Clinical statistics do not support this method. Intrat[illegible] injection necessitates disturbance of patient at a ti[illegible] *complete rest is the first essential of treatment.*

CONCLUSION from present evidence.—Subcut[illegible] or intramuscular methods should never be o[illegible] onset, an intravenous injection may be given [illegible] circulating toxin.

ANAPHYLAXIS.—*See* DIPHTHERIA.

b. DOSAGE.—Statistics have as yet given little [illegible] correct dosage. Probably amounts given, [illegible] present, are far too small. Use high potency [illegible] when possible. Dosage here recommended: at 30,000 units on first day by subcutaneous or [illegible] muscular methods, and aim at 50,000; maintain 2[illegible] to 30,000 units daily for three to four days; if symptoms improve, dosage reduced rapidly. By intravenous injection, 30,000 units may be readily given, but not to be deducted from above.

MORTALITY.—As the war progressed, *the incubation period* lengthened and the mortality fell. In hospitals in England mortality fell from 57·7 to 28·3 per cent in 1917 (Bruce). In France, change less marked among the severely wounded. In comparative period 1916, mortality in England 36·5 (200 cases) and in France 73·7 (160 cases), mean being 53 per cent. Improvement due to: (1) Prophylactic injections; (2) Better treatment of wounds. Unfortunately, no statistics justify conclusion that treatment with serum has diminished the mortality.

Symptomatology.—

INCUBATION PERIOD.—Very variable. *Commonest eight to*

twelve days. Very rare under five days: never under forty-eight hours. Upper limit doubtful; definite cases of 100 to 200 days. (*See above*, RESULTS OF PROPHYLACTIC INOCULATIONS.)

PREMONITORY SYMPTOMS (rarely observed except with prophylactic injections).—Rigidity, twitching, irritability, spasms and pains in muscles near wound, especially flexors.

SYMPTOMS.—Characterized by the development of tonic spasm of muscles, with frequent paroxysms.

INITIAL SYMPTOMS.—May be slight sore throat, difficulty in swallowing, and stiffness of neck.

ONSET OF DEFINITE SPASM.—(1) *Masseters* and muscles of mastication. Often noted first on waking. (2) *Muscles of back of neck. Spasm* extends in order to (3) Abdominal muscles, especially recti; (4) Back; (5) Limbs.

Concomitant symptoms commonly are: (*a*) Profuse sweating; (*b*) Rise of temperature; (*c*) Rigidity of abdomen.

CONDITION DEVELOPED.—Tonic spasm and rigidity of muscles produce characteristic phenomena: (i) *Trismus*: severe spasm of muscles of mastication, teeth clenched, difficulty in feeding increased by spasm of pharyngeal muscles. Unable to open mouth or speak. (ii) *Risus sardonicus*: lips stretched over closed teeth in ghastly smile. (iii) Eyes partly closed: forehead wrinkled. (iv) Head retracted to varying degree. Back may also be bent (opisthotonos). (v) Lower extremities usually extended, very stiff: knees sometimes flexed. (vi) Elbows may be flexed. *Hands usually escape.* (vii) Abdomen very rigid.

Paroxysmal exacerbations of spasms, usually with agonizing pain, occur as result of stimuli, e.g., movements, sudden noises, or apparently spontaneously.

Pulse usually rapid, 100 to 120.

PROGRESS IN FAVOURABLE CASES.—Paroxysms diminish in severity and frequency: tonic spasm slowly passes away.

PROGRESS IN UNFAVOURABLE CASES.—*Paroxysms and rigidity increase in severity.* Pulse often very rapid. *Temperature* often high but irregular: occasionally low. *Urine* may contain acetone bodies, albumin, and casts.

Death may occur from (1) Exhaustion: in these the spasm may have continued several days without alteration: starvation a probable factor. (2) Asphyxia: spasm of respiratory muscles and glottis. (3) Cardiac failure: pulse very rapid.

Mental condition may remain clear: usually (and properly) obscured by sedatives.

Duration: death usually within seven days: uncommon after ten days.

SEQUELÆ.—Affected muscles may remain stiff for long periods, especially jaw muscles. *Recurrences* are on record, following shortly after apparent recovery. No other complications.

Prognosis.—Varies with:—

(1) LENGTH OF INCUBATION PERIOD.—Improving in general as

Tetanus—Prognosis, *continued.*

period lengthens; but specially marked in contrasting durations over and under eleven days. Approximate mortality: ten days and under, 60 to 70 per cent, eleven days and over, 40 to 50 per cent.

2. RAPIDITY OF SPREAD of stiffness and spasms, and also frequency and severity of spasms.
3. SEVERITY OF WOUND. (Site of wound of little influence.)
4. HYPERPYREXIA, AND VERY RAPID OR IRREGULAR PULSE, are serious signs.

Mortality of all cases, 45 to 70 per cent. (*See also* TETANUS DURING THE WAR, and LOCALIZED TETANUS.)

Diagnosis. Onset in jaw and posterior neck muscles: note also sweating, early abdominal rigidity, and rise of temperature (rarely absent).

TRISMUS.—Reflex from teeth, Vincent's angina, tonsillitis, etc., or osteo-arthritis of jaw. No rigidity of neck muscles or very slight. Difficulty rare

STRYCHNINE POISONING.—(1) Jaw and neck not specially affected; (2) Complete relaxation between spasms, (3) Temperature normal.

TETANY.—(1) Rare in adults, (2) Extremities mainly affected with characteristic posture; (3) Gastro-intestinal disturbance.

HYDROPHOBIA.—Psychical disturbances prominent. Spasms specially affect larynx.

HYSTERIA.—Nervous wounded men with knowledge of symptoms occasionally develop trismus: other symptoms absent

BACTERIOLOGICAL METHODS.—Inoculation of mice reliable. Never delay treatment to await result.

Localized Tetanus.—Occurrence practically confined to the War (except cephalic type). Comparatively infrequent as result of prophylactic injections of horse serum, probably preventing generalized tetanus but not completely neutralizing toxin.

INCUBATION PERIOD usually very long, many weeks.

ONSET with stiffness near wound: slight spasms follow: finally may be extreme chronic rigidity. In rare cases becomes generalized, and all intermediate forms occur.

PROGNOSIS.—Death very rare when spasm remains localized.

TREATMENT.—For first two or three day or two full treatment of tetanus, but rapidly relaxed, especially serum, when condition remains localized.

CEPHALIC TETANUS.—Mortality 77 to 78. Occurs only in wounds of head and neck. Spasm of masseter muscles of face, and usually of pharynx, with facial paralysis. The facial paralysis is unilateral: rarely bilateral, and rarely absent. Almost invariably fatal.

Always rare (16 in 1917), but great rarity during the War was noticeable.

Treatment.—*Rest and quiet, sedatives, and food* are the essentials of treatment. Onset of tetanus is not an indication, *per se*, for further surgical treatment of wound.

REST AND QUIET.—Isolated in a darkened room. All noise, disturbance of patient, and examination to be reduced to an

absolute minimum. Shoulders well raised to aid respiration and relax abdominal muscles. Head supported.

SEDATIVES.—Should never be omitted. Chloral hydrate or chloretone as basis: bromides alone of little effect. Chloral hydrate gr. xv to xx, with potassium bromide gr. xx to xxx: 4-to 6-hourly by mouth. Only by rectum (double dose) when mouth impossible. Morphia is of less value.

Chloroform anæsthesia is sometimes employed when spasms very severe.

FOOD.—Peptonized milk with beaten-up eggs. Three pints of milk daily and 4 to 6 eggs: larger amounts if possible. Brandy added (ʒiv to ℥vj daily) if pulse very rapid or irregular. Feed by tube through teeth or nasal tube, if unable to swallow. Rectal feeding only as absolutely last resort, owing to disturbance of patient and deficient absorption from rectum: give glucose (5 per cent) and alcohol only (*see* GASTRIC ULCER).

ANTITETANIC SERUM.—(*See* pp. 105, 107.)

Note.—Injections of carbolic acid near the wound, and intrathecal injections of magnesium sulphate, did not prove of value during the War.

There is no method of treatment of any repute, even entirely without serum, which cannot be upheld from the literature by [illegible] cases treated practically or entirely "without a death".

CHAPTER XV.

GLANDERS.

[illegible] acute or chronic infectious disease due to *B. mallei*, and primarily affecting horses and asses. Characterized in man by inflammatory and suppurative lesions arising especially in nasal mucous membrane and subcutaneous tissues, and occurring in an acute and a chronic form.

Bacteriology.—Bacillus discovered by Loeffler and Schutz in 1882. Isolated from man by Weichselbaum in 1885.

MORPHOLOGY.—A non-motile, non-sporing bacillus, in shape resembling tubercle bacillus, but thicker: is often beaded. *Stains* with ordinary stains: *Gram-negative*. *In tissues*, mainly extracellular: numerous in acute and scanty in chronic forms.

CULTURAL CHARACTERISTICS.—Grows readily on ordinary media: best on blood serum or potato at [illegible]° C. Growth visible in two days. On potato, a yellowish growth, which by eighth day becomes a characteristic chocolate colour. Easily killed, except by drying.

GLANDERS IN ANIMALS.—Horses, asses, and mules especially affected. Cattle immune. Occurs in two forms: (*a*) Glanders, involving nasal mucous membrane; (*b*) Farcy, involving the lymphatics.

Glanders—Bacteriology, *continued.*

MODE OF INFECTION IN MAN Is a rare disease Infection occurs by direct contagion from a diseased animal, the bacilli being discharged from the nostrils or from sores Bacilli may enter the human being through nasal mucous membrane or abrasion of skin Laboratory infection among experimenters occurs with exceptional readiness, and many deaths are on record Infection from patients also common, and extreme care necessary

Morbid Anatomy.- In acute forms lesions show ordinary suppurative changes In chronic forms, an early glanders nodule resembles a tubercle, with greater acute inflammatory changes and less proliferation Glanders is regarded as an infective granuloma

Symptoms. Glanders in man occurs in two forms acute and chronic

1 ACUTE GLANDERS *Incubation period* usually one to five days *Onset* with (*a*) general malaise, (*b*) redness, swelling, and lymphangitis at site of inoculation Constitutional symptoms and evidences of general infection in 2 or 3 days

ERUPTION OF PAPULES Especially on face and joints rapidly becoming pustular, as in small pox.

NASAL MUCOSA —Nodules form, ulcerate, and discharge with subsequent necrosis and foul discharge, nose becomes extremely swollen and red

ABSCESSES form. subcutaneous or muscular Often within forty eight hours certain tissues become phlegmonous fever high

BRONCHITIS. Common, and frequently pneumonia from foci in lungs. Typhoid state may occur, with marked intestinal symptoms

EXTREME COLLAPSE and ACUTE SEPTICÆMIA follow

DEATH in from one to three weeks Mortality 95 per cent

Albuminuria usually present Secondary infections common, but lymphatic glands and testes not specially affected in man The abscesses are the characteristic feature; the eruption and nasal symptoms may be absent These cases are sometimes described as 'acute farcy'.

2. CHRONIC GLANDERS - *Incubation period* 10 days or upwards At *onset* may be rash, papular, pustular, vesicular or erysipelatous

FORMATION OF ABSCESSES is characteristic symptom, subcutaneous and intramuscular, especially near joints. Abscess ruptures irregular ulcer, paper results discharge often very offensive. Abscesses often heal, and with great frequency break down again, or fresh abscesses form. Condition *often extremely chronic*: may be latent for months or years and then relapse and fresh abscesses form. *Recovery* in 50 per cent of cases, but at any stage, even after apparent cure, condition may develop symptoms of acute form and be fatal. In chronic condition, nose and lungs usually escape.

Diagnosis.—

OCCUPATION —Often suggestive.

CLINICAL DIAGNOSIS —Extremely difficult

BACILLI —These may be present in discharges, and recognized in films and cultures Occasionally isolated from blood cultures

INOCULATION INTO ANIMALS —Intraperitoneal injection in guinea pigs results in *suppuration of testes* in 2 to 3 days Inoculation may be made from cultures or direct from discharge, but in latter case secondary pyogenic organisms may cause acute peritonitis rapidly

INJECTION OF MALLEIN —Is of great value *diagnostically* for animals but untried in man Mode of preparation and technique resembles tuberculin Precipitin, agglutination and complement fixation tests are also of value in animals

Treatment.—

PROPHYLAXIS —Glandered animals must be destroyed, and premises thoroughly disinfected Attendants on patients must be warned of the danger of infection Soiled linen etc , should, if possible be destroyed otherwise, carefully boiled

ACUTE CASES Treatment is symptomatic only

CHRONIC CASES —All abscesses should be opened as they occur, and treated with antiseptics No drug appears to have any special action, but sodium benzoate is recommended

Vaccines have been tried and some good results recorded

CHAPTER XVI

ANTHRAX.

(*Malignant Pustule Wool sorters' Disease Splenic Fever* in animals)

An acute infectious disease, caused by *B anthracis*, occurring in man in a cutaneous form as malignant pustule , in a pulmonary form as wool-sorters' disease , and very rarely in an intestinal form.

Etiology.—Primarily a disease of animals, especially sheep and cattle, causing a septicæmia with enlarged spleen and pulmonary congestion. Occurrence is world wide, most frequent in Russia and France Organism was discovered by Pollender in 1849, and investigated chiefly by Davaine, Koch, and Pasteur.

Bacteriology.—

MORPHOLOGY —A large rod shaped bacillus with clear cut ends, length 6 μ and upwards. Non motile. Forms spores readily. Bacilli in cultures often joined end to end in a chain. Stains with ordinary stains, and is Gram-positive. Often a capsule.

SPORES AND SPORE FORMATION.—Never present in living tissues. Probably due to absence of free oxygen. Form readily in media and are always present in cultures Especially frequent when organism is under slightly adverse conditions, e g , lying on soil or in dead animals. Spores are seen in body of bacillus

Anthrax—Bacteriology, *continued.*

or lying free. Stain with weak carbol-fuchsin, while body o bacillus may be stained by methylene blue.

Extremely resistant. When dry, alive after a year. Withstand boiling for 5 minutes. Very resistant to dry heat: also to gastric juice.

CULTURAL CHARACTERISTICS.—Grows readily on all ordinary media. Most characteristic are: On agar plates at 37°, in 12 hours, colonies visible with wavy outline like locks of hair. In broth forms long spiral threads. In deep gelatin stab, radiating spikelets and slow liquefaction, commencing at surface. Bacillus not very resistant apart from spores.

Filtered cultures non-toxic.

Anthrax in Animals.—Condition varies in severity, but is a septicæmia characterized by bloody mucous discharge from nose and mouth, the sanious discharges containing numerous bacilli. Death in twelve to forty-eight hours.

MORBID ANATOMY. *Spleen* greatly enlarged. Lymphatic glands enlarged. Lungs congested. Cloudy swelling in all organs. Bacilli are present everywhere, especially in spleen, in capillaries, and lymphatics.

SUSCEPTIBILITY.—Varies greatly in different species. The large herbivora, sheep and cattle, highly susceptible, although certain Algerian sheep are immune. Adult carnivora and white rats are immune.

Man has considerable immunity.

MODE OF INFECTION. Numerous bacilli are deposited from the mucous discharges and form spores; hence a pasture may remain infective for years and the spores be scattered by wind and water. The spores pass through the stomach, resisting the gastric juice, and thus animals are attacked from the intestines. Buried carcases are a possible source of infection. Pasteur believed that earthworms may thus be a factor, but Koch disproved it.

PASTEUR'S METHOD OF IMMUNIZATION. Pasteur attenuated cultures by growth at 42°, and immunized animals by inoculation. The method is practised on a very extensive scale, and is of extreme value.

Anthrax in Man.—Almost confined to workers in hides, hair, and foreign wools; very rarely in butchers; occasionally from infected shaving brushes. The clinical symptoms vary according to mode of inoculation, external or internal. The following varieties are usually described:—

(1) *Malignant pustule*, or cutaneous anthrax. An erysipelatous anthrax, or anthrax œdema, also occurs rarely. (2) Pulmonary anthrax, or wool-sorters' disease. (3) Gastro-intestinal anthrax, or mycosis intestinalis; rare. Malignant pustule forms 95 per cent of all cases.

1. MALIGNANT PUSTULE.—Site of inoculation most commonly face, back of neck, and arms; being rubbed by hides carried on back. In a few hours, *itching* at site of inoculation. *Papule* forms in one to three days: rapidly becomes a vesicle containing

clear or bloody fluid and surrounded by area of congestion: *central necrosis* occurs Typical *malignant pustule* present in 1½ to 3 days—viz, central black eschar surrounded by a ring of vesicles, and outside this an area of congestion The pustule never contains pus Subcutaneous œdema spreads from the pustule Lymphatic glands in area swell

GENERAL SYMPTOMS Slight in early stage, but in absence of recognition and of removal of pustule rapidly become severe, with malaise, faintness, weak pulse, and collapse *Temperature* is high Severity of general symptoms is out of proportion to size of local lesion Pain usually slight Septicæmia develops as in internal forms, but modified Death occurs in three to five days in absence of treatment The mind is usually clear to the end Cases vary in severity Eschar may slough out, and recovery occur without treatment

MORTALITY Varies with position of pustule Most fatal on face, 25 per cent On lower limb, 5 per cent Post mortem the spleen is lightly enlarged and few bacilli present in organs Mortality low with early treatment

ANTHRAX ŒDEMA No pustule occurs Infection possibly from hair follicle Œdema commonly commences on eyelid and spreads rapidly Rarely diagnosed, always fatal Rare

PULMONARY ANTHRAX OR WOOL SORTERS' DISEASE — Infection occurs through the lungs Onset rapid Rigor, rapid respiration, pain in chest, rapid and feeble pulse Cough and bronchitis usual *Temperature* high Œdema of chest wall develops of gelatinous consistency Much frothy mucus Extreme collapse and death in one to three days Mind usually remains clear

PROGNOSIS Improves with longer duration In some cases marked cerebral symptoms convulsions delirium etc due to bacilli in capillaries of brain Diarrhœa occasionally severe Recovery extremely rare

MORBID ANATOMY Main lesion in trachea and large bronchi, with œdema and hæmorrhages Lungs œdematous Pleural and pericardial effusions Great enlargement of thoracic glands Apart from thorax, changes in the organs slight Bacilli are numerous in the affected sites, but scanty or absent in the spleen and other organs

3 INTESTINAL FORM —A few outbreaks have occurred abroad, probably from diseased flesh Resembles acute food poisoning. Chill, vomiting and diarrhœa, convulsions, enlarged spleen

Diagnosis. —

MALIGNANT PUSTULE —Diagnostic features are (1) Occupation. (2) Appearance of pustule. Rapid onset, eschar, œdema, no pus, no pain. (3) Severity of general symptoms as compared with local lesion. (4) *Bacteriology.* Bacilli are present in edge of eschar, and in cultures Inoculation of cultures or material from pustule

Anthrax—Diagnosis, *continued.*

into guinea-pig causes malignant gelatinous œdema at site o inoculation, with hæmorrhages into organs, and bacilli present in large numbers, especially in capillaries.

DIAGNOSIS from *chancre* by rapid onset, from *cellulitis and erysipelas* by absence of pain; from *boils* by absence of pus; from *malignant œdema* (no gaseous crepitations), from *glanders* (no nasal discharge and no red vesicles).

PULMONARY ANTHRAX.—In early stages usually impossible.

Treatment.—

MALIGNANT PUSTULE.—(1) Excise freely. (2) Inject Sclavo's serum, 40 c.c. subcutaneously, injecting not more than 10 c.c. at each site. Repeat in twelve hours if necessary.

Injection of a few minims 1–40 carbolic acid at several places near pustule has been recommended.

PULMONARY ANTHRAX.—No local treatment is of value.

CHAPTER XVII.

LEPROSY.

An infective disease of marked chronicity caused by *B. lepræ*, and characterized by lesions in the skin and mucous membranes or in the nerves, and in advanced cases frequently in both.

History and Geographical Distribution. Is referred to in most ancient literature of the East, though probably other diseases were included. Is most prevalent in the tropics, but distribution is not limited geographically. Occurs in Norway and Iceland. Most frequent in India and China. In South Africa has recently increased. Formerly spread over the entire Old World, but commenced to decline in the 15th century. In Great Britain now only imported cases. Did not occur in America in pre-Columbian days.

Bacteriology.—The *B. lepræ* was discovered by Hansen in 1871. Slender, non-motile bacillus, resembling tubercle bacillus in appearance and staining reactions. Is Gram-positive and acid-fast (in 12 per cent acid). Stains with ordinary stains more readily than tubercle bacillus. Bacillus has never been cultivated satisfactorily. Possibly it is really a non acid-fast streptothrix. Animals cannot be infected.

Morbid Anatomy.—The lesion is a granuloma. *The leprous nodule*, in any site, consists of granulomatous tissue with endothelioid cells of various sizes. Enormous masses of bacilli are present,

mainly within cells The so called *lepra cells* contain numerous bacilli often arranged parallel Some of these cells are probably lymphatics with thrombi of bacilli Giant cells may be present Caseation does not occur, unless tuberculosis is also present

Terminally, tissues affected are skin, mucous membranes, and nerves, also liver, spleen, and testes

Mode of Infection. *B lepræ* does not fulfil Koch's postulates but is accepted as cause of leprosy It has never been found outside the human body, and therefore infection, apparently, must be conveyed from a leper The slow progress of the disease and immunity of animals have rendered investigation of modes of infection difficult and nothing definite is yet known Possible methods are

1 INOCULATION There is no evidence that biting or other insects can convey infection Results of direct inoculation experiments in man are doubtful

HEREDITY – Has very slight if any, influence No new born infant is leprous and cases rare under 5 years Several members of a family may be attacked but are usually exposed to possibility of a common infection Hansen found that of the descendants of 160 Norwegian lepers who emigrated to America none were leprous

3 BY CONTAGION The nasal mucosa is early infected, and numerous *B lepræ* are present in the discharge Infection may thus result by inhalation though the lungs are rarely affected The bacilli are also present in discharge from sores This is most probable mode of infection but contagiousness must be very low Doctors and attendants extremely rarely infected An additional factor may be necessary such as an insect Hutchinson considered that eating salt or stale fish induced infection but Lapians never eat fish and are very subject to leprosy Bad hygiene undoubtedly increases pr[illegible]

Varieties of Leprosy. Two main groups

1 NODULAR LEPROSY Also called *tubercular* or *tuberose leprosy* Characterized by pyrexial attacks, and granulomata of skin and mucous membranes

2 ANÆSTHETIC LEPROSY Also called *maculo anæsthetic, nerve,* or *atrophic leprosy* Characterized by macules and nerve changes

'MIXED' FORMS are common These may –

i Commence as 'nodular' and develop symptoms of 'anæsthetic' type Very frequent

ii Develop both symptoms together Less frequent

iii Commence as 'anæsthetic' and develop symptoms of 'nodular' Uncommon

Symptoms.—

INCUBATION PERIOD —Many years

NODULAR LEPROSY —

PRODROMAL SYMPTOMS Occasional pyrexia and malaise.

Leprosy—Symptoms, *continued.*

FIRST STAGE.—Attacks of fever, with swelling or erythema of face. Fever subsides and a patch of erythema remains. Several attacks yearly for one to two years.

SECOND STAGE.—Repeated attacks of fever. Patches swell and become infiltrated. Usually hyperæsthetic. The 'tubercles' commence in the patches, at first as papules. They multiply, grow, coalesce, and form the typical flat masses of leprotic tissue. Masses become anæsthetic.

SITES ATTACKED.- Usual order: face (especially lobes of ear), then forearm, limbs, thighs, buttocks. Mucous membranes, especially nasal, as early as face.

ATTACKS OF FEVER (leprotic fever). Duration varies, often one to two weeks. Frequently 102° to 103°. Rarely no pyrexia.

FULLY DEVELOPED.—Marked changes are:

Face. -Natural lines obliterated and replaced by creases between masses of growth. Hair on face drops out, but scalp not affected. General expression sombre and 'leonine' Ears, especially lobes, much thickened.

Mucous membranes.—Nasal discharge Nose flattened. Pharynx and larynx affected. Vocal cords fixed voice hoarse, or only whisper. Tongue infiltrated or ulcerated. Lips cicatrized and stenosed.

Limbs.- Covered with nodules and masses to varying degrees.

Eyes Affected commonly Conjunctivitis, keratitis, etc.

SUBSEQUENT PROGRESS. Variable (1) Quiescent for many years, or marked remissions. (2) Exacerbations, more common. Pyrexial attacks occur, with spread of growth. Ulceration of masses common, with discharge cicatrix on healing. often chronic. (3) 'Mixed' form frequently, develops, with symptoms of 'anæsthetic' leprosy.

ANÆSTHETIC LEPROSY.—

ONSET. -Insidious. Progress very slow.

PRODROMAL SYMPTOMS. Indefinite. malaise and chills, vague pains, hyperæsthesia, or deafness.

FIRST STAGE.—*Maculæ* are first sign, one or several Diameter 1 to 2 inches. Areas of (1) *erythema*, (2) increased pigmentation, or (3) decreased pigmentation Not raised Sensation normal. Sweat glands of area affected, and patches are dry even after pilocarpine. Erythema of brown tinge in white races and light in coloured races. Fresh maculæ appear, sometimes in relation to a peripheral nerve

Site.—Back and buttocks most common; face uncommon. *Ulnar nerve* may be palpable at elbow in earliest stages.

SECOND STAGE.—*Maculæ* spread. Centre often fades and periphery extends and coalesces with others. Large area affected. Face often discoloured, but never 'white as snow'. *Areas anæsthetic*, losing touch, heat and cold, and pain, in order.

Nerve trunks thickened: ulnar, then median, posterior tibial, and peroneal. Hence: (1) Anæsthesia of extremities, extending; (2) Contractions, especially of 4th and 5th fingers.

THIRD STAGE.—*Eruption* inactive: may fade. *Nerve lesions extend*; in rare cases become quiescent.

FULLY DEVELOPED.—

Skin.—Dry and parchment-like. *Anæsthesia extreme.*

Contractions.—Ulnar nerve especially affected, whence 'claw hand'.

Trophic changes.—(1) Perforating ulcers, arising from bullæ or injuries resulting from anæsthesia. (2) Loss of fingers, toes, or more extensive parts from necrosis or interstitial absorption of bone, or from gangrene or suppuration.

Eyes.—Affections result from lesions of 5th and 7th nerves, but not frequent.

Occasionally 'nodular' leprosy also develops.

Diagnosis.—Advanced cases easy.

Early Nodular Leprosy.—Bacilli present (1) in nasal secretion, (2) in excised piece of skin. Clinical diagnosis from syphilis, tuberculides, erysipelatoid attacks from septic foci. Wassermann reaction is often positive in leprosy.

Early Anæsthetic Leprosy.—Diagnosis depends on maculæ, thickened nerves, and anæsthesia. Often no bacilli in nose or skin.

Prognosis. Either form may become arrested, especially when patients from the tropics are kept in cool climates. In usual condition, disease progresses over twenty, thirty, or more years. Death follows from nephritis, tuberculosis, or progressive exhaustion.

Treatment.—

GENERAL TREATMENT. Diet and cleanliness of greatest importance. Europeans must not return to the tropics.

LOCAL TREATMENT. Finsen light, or x rays.

DRUGS.—*Chaulmoogra oil* internally for long periods: commence ♏v, t.d.s., and increase to ʒij. Intramuscular injections of 1 to 3 c.c. weekly of following mixture: chaulmoogra oil 60 c.c., camphorated oil 60 c.c., resorcin 4 grm. (Heisser). Gynocardate of soda (from fatty acids of chaulmoogra oil) intravenously, gr. $\frac{1}{10}$ to $\frac{1}{4}$ in 2 per cent saline solution and 0·5 per cent phenol (Rogers); larger doses also given intramuscularly and orally. Ethyl ester chaulmoograte: intramuscularly, 1 c.c. increasing to 5 c.c. The results of recent methods are encouraging, especially the last.

PROPHYLAXIS.—Segregation and isolation are unnecessary when sanitary conditions exist in the home.

CHAPTER XVIII.

TUBERCULOSIS.

I. GENERAL FEATURES, ETIOLOGY, AND HISTOLOGY.

History.—Pulmonary tuberculosis was known to the Greeks. Considered as contagious by Hippocrates and Galen, and generally so believed until early in the nineteenth century.

SYLVIUS, 17th century, described the tuberculous nodule and its relationship with phthisis, and considered the nodule similar to scrofulous glands. MORTON in same period also described the nodule.

LAENNEC, 1819, traced changes from tubercles to caseation, ascribed all forms to tuberculosis, upholding the 'unity of tuberculosis', and discovered the physical signs, but unfortunately, considered it non-contagious. View became widespread that condition depended on special diathesis.

VIRCHOW opposed the 'unity of tuberculous lesions', considered that scrofula and tuberculosis were independent, and believed that ordinary inflammatory lesions might end in tuberculous caseation.

VILLEMIN, 1868, experimentally reproduced tuberculosis in animals, thus proving contagious nature. These researches were widely discussed but their conclusiveness by no means recognized. At this period, the morbid anatomy and histology was carefully studied.

COHNHEIM AND SALOMONSEN, 1879, injected tuberculous matter into the anterior chamber of the eye of guinea-pigs and rabbits, tuberculous nodules resulting and, later, disease of lymphatic glands and finally acute tuberculosis. These experiments were widely accepted as proof of contagion. Search for the causal bacterium was now in progress.

KOCH, 1882, announced discovery of *B. tuberculosis* and by isolation, cultivation, and inoculation into animals finally proved its contagiousness and the tuberculous nature of many lesions. The contagious theory, thus proved, temporarily obscured the importance of diathesis and of other factors.

EHRLICH, immediately on publication of the above, discovered the acid-fast method of staining which, with slight modification, is known as Ziehl-Neelsen's method.

KOCH, 1889, reported the preparation of *tuberculin*, for which curative powers were claimed. Koch did not consider that his investigations were complete, and published them prematurely under pressure.

KOCH, 1901, made the statement that human and bovine tuberculosis were independent, and that man could not be infected from animals. Now disproved after lengthy experiments.

Bacteriology.—

B. tuberculosis is the essential cause.

MORPHOLOGY.—*Thin rods*, straight or slightly bent: *beading often present*. Ends may be thickened. In tissues scattered or frequently in small clumps. Filaments and aberrant forms in old cultures.

GROWTH IN CULTURE.—None on ordinary media. On Koch's inspissated blood serum appears about fourteenth day, forms dry scales. Subcultures grow on glycerin agar. Best on Dorset's egg medium. For tuberculin grown in glycerin broth.

STAINING REACTIONS.—Affected by presence of a fatty capsule. With ordinary stains, very slow. Best stained by Ziehl-Neelsen's carbol-fuchsin method, being 'acid-fast' (and also 'alcohol-fast', thus differing from smegma bacillus). Gram-positive, but stains very slowly.

RESISTANCE.—Marked. Virulent in dried sputum after two months. Killed by 100° C. in fluids and tissues, but virulent after an hour if dry.

OCCURRENCE OF THE BACILLUS IN THE BODY.—

IN ACUTE LESIONS. Often numerous, especially with rapid caseation. Numerous in spleen in acute tuberculosis in children. Present, though less numerous, in urine, cerebrospinal fluid, and fæces, in tuberculosis of respective systems: in pus, when caseation rapid. In acute miliary tuberculosis, rarely numerous.

IN CHRONIC LESIONS.—Very scanty, e.g., in pleural effusions, caseous matter, lymphatic glands. Animal inoculation often necessary for proof of presence. Bacilli usually extracellular occasionally a few in giant cells, leucocytes, and epithelioid cells. In cattle, in general more numerous and commonly in giant cells.

IN BLOOD.—Isolated by culture by Rosenow repeatedly; rarely found by other observers.

OUTSIDE THE BODY.—Chiefly present in milk. Isolated from dusts of streets, etc., but often absent, even in sanatoria.

VARIETIES OF B. TUBERCULOSIS.—Four principal types: (1) Human; (2) Bovine; (3) Avian; (4) Piscine. Koch, 1901, stated that bovine and human tuberculosis were distinct, and could not be transmitted from one to the other: based on (*a*) difficulty of infecting cattle with human bacillus, (*b*) asserted rarity of primary intestinal tuberculosis in man.

ROYAL COMMISSION CONCLUSIONS, 1912.—Two main types of bacillus, human and bovine, differing in:—

1. CULTURE.—Human: growth abundant (eugonic), dry, scaly, and yellowish. Bovine: bacillus shorter and thicker; growth scanty (dysgonic), especially on glycerin media, moist, white, and smooth; vitality less.
2. VIRULENCE.—Bovine more virulent to animals. Inoculated into cattle, bovine causes fatal general tuberculosis; human, a local lesion only. To rabbits, bovine is fatal and human non-virulent. Both virulent to guinea-pigs.

Tuberculosis—Bacteriology, *continued.*

3. DISTRIBUTION.—Cattle: always bovine bacillus. Man: in bone, joint, and primary abdominal tuberculosis in children, and in lupus, nearly 50 per cent bovine; adult pulmonary disease almost always human type.
   No proof that bovine changes to human type in the body, but still in dispute.

CONCLUSIONS: (1) Infection in phthisis is of human origin, with rare exceptions; (2) In other forms mentioned infection is equally of human and of bovine origin (milk).

OTHER TYPES OF TUBERCLE BACILLI.

Avian.—Birds, including fowls, are immune to human type. Avian type is not found in man. Rabbits and mice alone of mammals are susceptible in any degree: guinea-pigs are immune. The bacillus grows more readily, with a moister surface, and at a higher temperature (43.5° C.), than the human type.

Piscine.—Morphologically resembles human type, but no growth above 26° C.; non-pathogenic to mammals.
   In pigs the type is nearly always bovine, rarely human or avian: lesions intestinal.

OTHER ACID-FAST BACILLI occur widely spread, e.g., in butter (Rabinowitch's bacillus), in milk, hay (Timothy-grass bacillus): may cause local lesions on injection. Also, in animals, Johne's bacillus (chronic bovine pseudotuberculous enteritis).
   Smegma bacillus: acid-fast but not alcohol-fast.

VARIETIES OF HUMAN BACILLUS.—Three forms are described by Much: (1) Ordinary acid-fast bacillus; (2) A fine form containing granules; (3) Free granules. The last two forms do not stain with Ziehl-Neelsen, not being acid-fast, but stain by the Gram-Much method: on inoculation they produce tuberculosis, acid-fast bacilli being present: are specially present in caseous matter. Much's results not yet satisfactorily confirmed.

Predisposing Causes.—The tubercle bacillus is almost universal. Post-mortems show some tuberculous lesion in very high percentage: by von Pirquet's reaction Hamburger estimates that by age of twelve 90 per cent of people have been infected. Hence predisposing causes are of vast importance, influencing result of an infection with tubercle bacilli. Predisposing influences may be inherited or acquired.

HEREDITY.—Tuberculous diathesis long recognized. Two types often described: (1) *Hippocrates' habitus phthisicus*: delicate skin, blue sclerotics, thin flat chest, winged scapulæ; (2) Scrofulous type: coarse skin, broad face and features, short heavy bones and build. Karl Pearson by statistical studies has shown importance of heredity.

AGE.—Occurs at all ages. Under 10 years, special tendency to bone, gland, joint, and other forms of tuberculosis. Above 10

years, pulmonary lesions commence to predominate. Deaths are highest from 18 to 35 years.

RACE.—Very fatal to negroes. Jews, low mortality.

ENVIRONMENT.—Of supreme importance. Bad ventilation, aided by spitting, insufficient exercise, and ancillary factors, accounts for mortality in poor districts, gaols, etc.; first by reducing physique, secondly by increasing frequency of infection.

OCCUPATION. Influences: (1) General, as in environment; (2) Special in certain occupations, e.g., 'grinders' rot' (*see* PNEUMONOCONIOSIS). In chest hospitals prevalence varies among attendants and nurses. Rare at Brompton Hospital.

RELATION TO OTHER MORBID CONDITIONS.—

✓ PREDISPOSING TO INFECTION or to spread of a latent focus:—

1. *Certain acute respiratory affections.* Not infrequent after influenza, measles, whooping-cough. Pneumonia does not predispose to tuberculosis: cases so terminating are tuberculous from onset: so also with pleurisy and bronchitis.
2. *Congenital morbus cordis.* Frequent as a termination, especially in pulmonary stenosis (lungs often small and undeveloped).
3. Diabetes, alcoholism, and debilitating diseases such as chronic nephritis, cirrhosis of liver: terminal phthisis frequent.
4. Typhoid fever. In two years subsequent to attack, mortality from phthisis trebled. (*See* p. 19)

✓ ANTAGONISTIC TO INFECTION:

1. Mitral stenosis. Rokitansky showed extreme rarity of association, and ascribed it to venous stasis (also next group). With other valvular lesions less unusual, but not common.
2. Deformities of chest from any cause, e.g., rickets.
3. Tuberculosis of bones and joints (to lung disease).
4. Gout. Association said to be rare.

PREGNANCY.—It is generally accepted that a woman with phthisis can pass with comparative safety through one pregnancy, and may with difficulty through a second, but that a third will prove fatal. Tuberculosis is often accelerated by pregnancy: progress may be rapid before or after parturition. Ascribed to changes in type of respiration and in blood-supply of lungs, to strain, and possibly to hyperglycæmia. Laryngeal tuberculosis is not uncommon and progresses rapidly. Except possibly in the earliest months, nothing is gained by abortion or premature labour. A tuberculous mother should never suckle an infant.

MARRIAGE.—A tuberculous subject should not marry for at least two years after the cessation of all symptoms of active tuberculosis.

CLIMATE.—Occurs in all climates, but rarer in dry high localities.

Tuberculosis—Predisposing Causes, *continued.*

TRAUMA.—Data inconclusive. Appears to predispose to tuberculosis of knee-joint. No proved influence on phthisis. (*See also* HÆMOPTYSIS.) Relation of head injuries to meningitis also unproved.

Sources of Infection.—(1) Sputum of phthisical persons. Danger mainly from droplets of sputum suspended in air on coughing or speaking. No ejection of bacilli on quiet breathing. (2) Milk of cattle with tuberculosis of the udder. In meat, bacilli are mainly killed by cooking.

Modes of Infection.—Four possible modes: (1) Heredity; (2) Cutaneous inoculation; (3) Inhalation; (4) Ingestion. The first two are of negligible practical importance.

HEREDITY.—Congenital tuberculosis extremely rare: ascribed to infection through placenta, which is usually affected. Infection by spermatozoon or ovum may be neglected.

CUTANEOUS INOCULATION. Occurs in butchers and post-mortem workers. Lesions usually remain local. Experimentally in animals may cause general infection. Lupus has followed vaccination.

INHALATION.—The main evidences are: (i) Tuberculous sputum supplies factor. (ii) The frequent onset in the lungs, some degree present in 60 to 100 per cent of all post mortems. (iii) Evidences of aerial contagion, e.g., spread in institutions and gaols. (iv) Frequency of phthisis in Japan, where no cows' milk is used. (v) Phthisis in adults is caused by the human type of bacillus (Royal Commission). (vi) Animal experiments.

Note.—Husband and wife infections are notoriously rare.

INGESTION.—Infection may occur through (*a*) tonsil, (*b*) alimentary canal. The main evidences are: (i) Presence of tubercle bacilli in cow's milk. (ii) Bone, joint, abdominal tuberculosis in children is due to bovine bacillus in 25 to 50 per cent (Royal Commission). (iii) Frequent infection of cervical glands ('scrofula'). (iv) Animal experiments.

Paths of Infection in Pulmonary Tuberculosis.

MAIN THEORIES:—

INHALATION.—(*a*) Direct to small bronchi, or (*b*) Bacilli penetrate tracheal mucous membrane, thence to tracheo-bronchial glands, thence by blood or lymph to lung tissue.

ORAL.—Through mucous membrane of mouth, pharynx, or tonsils (without causing lesions) to cervical glands: thence (*a*) To supraclavicular glands and to apex of lung, or (*b*) To bronchial glands first.

INGESTION.—(*a*) Through mucous membrane of intestine to glands, thence by thoracic duct and blood to lungs; mesenteric glands may or may not be affected. Or (*b*) Primary intestinal tuberculosis (rare), and spread thence.

Inhalation theory accepted until BEHRING, 1913, asserted that phthisis resulted from bacilli ingested in milk in childhood, bacilli passing through intestine and remaining latent.

Evidence is based on (1) Animal experiments; (2) Morbid anatomy.

ANIMAL EXPERIMENTS. VILLEMIN, 1868, KOCH, 1884, produced pulmonary tuberculosis by inhalation. FINDEL, 1907, showed that minute doses are pathogenic by inhalation but much larger doses cause no effect on ingestion: confirmed by many observers. Inoculation into animals proves that tonsils may be tuberculous without obvious lesions, supporting this path to cervical glands. Of ingestion experiments, CALMETTE and others showed (*a*) Ingestion may result in pulmonary tuberculosis; (*b*) The intestine and even mesenteric glands may show no gross change. BARTELS says that such glands are in a 'lymphoid' pre-tuberculous condition, producing tuberculosis on inoculation. Localization in lung attributed to preference of bacillus for lung tissue.

Conclusions.—(1) A small dose by inhalation is pathogenic; (2) By ingestion of large doses pulmonary tuberculosis may result, even without intestinal lesions.

MORBID ANATOMY IN MAN. *Apical Lesions*: In very early lesions Schmorl and Birch-Hirschfeld find onset in the smallest bronchi, causing a peribronchitis: thus supporting inhalation path.

GENERAL CONCLUSIONS.—

INHALATION. *The predominant path for phthisis.* Principal evidence: (1) Minute doses by inhalation experimentally produce pulmonary tuberculosis; (2) In man, human type of bacillus almost invariably present in phthisis; (3) Onset as peribronchiolitis.

INGESTION.—*Frequent or predominant path for bone, joint, and abdominal tuberculosis.* Principal evidence: (1) Presence of bacilli in milk; (2) In 25 to 50 per cent. bovine strain present. Possible but rare path for phthisis.

ORAL (TONSILLAR) PATH. Path for cervical glands. Subsequent path of bacilli and influence on phthisis uncertain.

Histology of Tuberculous Lesions.—The tubercle bacillus causes a chronic inflammatory change, *a granuloma*. The typical element is the 'tubercle': this is histologically identical with certain other local chronic inflammations, e.g., actinomycosis.

THE ELEMENTARY 'TUBERCLE'.—On arrival and multiplication of tubercle bacilli the following changes occur: (1) Fixed connective-tissue cells multiply, forming *epithelioid cells*; (2) Polynuclear leucocytes arrive, are destroyed, and are followed by small lymphocytes; (3) Giant cells may form; (4) A fibrillated reticulum may surround cells. A *giant-cell system* thus forms.

GIANT-CELL SYSTEM.—Features: (i) Giant cells near centre; (ii) Some caseation; (iii) A ring of epithelioid cells; (iv) An outer ring of small, mononuclear cells; (v) System is non-vascular; (vi) Tubercle bacilli amongst cells, but scanty;

Tuberculosis—Histology of Tuberculous Lesions, *continued.*

(vii) Often an outer zone of hyperæmia. *Rarely seen typically except when lesion very acute.*

Epithelioid Cells.—Large oval cells with oval faintly-staining nuclei and considerable protoplasm. May contain bacilli.

Giant Cells.—Formed by fusion of several epithelioid cells, or by multiplication of nuclei. Many nuclei gathered together at one end or edge. Rarely contain bacilli (but commonly so in cattle).

Variations.—Giant cells often absent. Epithelioid cells or mononuclear cells are sometimes absent.

GROWTH OF THE TUBERCLE.—

MILIARY OR GRAY TUBERCLE.—By fusion of several elements. *Size of pin's-head, semi-translucent, gray, firm, and projecting.*

YELLOW TUBERCLE.—*Gray tubercle increases by fusion with others,* caseation occurring simultaneously: thus forms yellow tubercle, an opaque yellow mass size of nut surrounded by ring of gray tubercles. Beyond is area of hyperæmia, and, in the lung, proliferated alveolar cells and small bronchi containing desquamated cells and exudation. A tubercle is always *non-vascular.*

SECONDARY DEGENERATIVE CHANGES.—(1) Caseation; (2) Fibrosis; (3) Calcification; (4) Softening.

CASEATION.—Commences in centre of tubercle, a coagulation necrosis, spreading outwards: cells stain badly, lose outline, and become débris: bacilli scanty or absent, but matter usually virulent on inoculation. Due to action of bacilli or their toxins.

FIBROSIS.—Commences at periphery: proliferation of connective-tissue cells: is result of inflammation set up by tubercle, an effort at repair. Caseation and fibrosis invariably occur. If fibrosis is successful, a capsule is formed and progress of tuberculosis checked, but bacilli in encapsulated caseous matter may be virulent years later if rupture occurs.

CALCIFICATION.—Caseous matter impregnated with lime salts, forming hard and harmless mass, e.g., a lung stone.

SOFTENING.—Caseous matter liquefied by exudation of fluid. Tends to occur near surface of body and where tissues are soft. 'Chronic abscess' results; contains white gritty sterile matter formed of cell débris, not true pus; wall of purplish granulation tissue loosely adherent to surroundings and containing tubercle bacilli.

DISTRIBUTION OF TUBERCLES IN THE BODY.—In adults: especially in lungs. In children: especially bones, joints, and lymph-glands. Rare in stomach, œsophagus, thyroid, and muscles, and unusual in pericardium.

METHODS OF EXTENSION IN THE BODY.—From a focus spread may occur by: (1) Mucous surfaces; thus sputum affects other parts of lung, or, after swallowing, the intestine. (2) Lymphatics. (3) Blood-stream: result may be (*a*) *local*, entering pulmonary artery and infecting region of lung, or (*b*) *general*, e.g.,

entering pulmonary vein and causing acute general miliary tuberculosis.

Distribution and Occurrence in Nature.—Widely distributed. Prevalent in man, cattle, and birds, especially fowls. Common in pigs. Occurs in fish. Rare in dogs, cats, sheep, goats, and horses. Not in rabbits or guinea-pigs, though both very susceptible to experimental inoculation. Common in confined monkeys.

INCIDENCE IN MAN.—Accounts for about one-seventh of deaths, but death-rate from tuberculosis has been falling for 50 years, especially in England, commencing before discovery of tubercle bacillus. Fall ascribed to: (1) Social improvement; (2) Earlier diagnosis; (3) Segregation in institutions.

II. MILIARY TUBERCULOSIS.

General miliary tuberculosis results when tubercle bacilli enter blood-stream from a primary focus, *e.g.*, an unencapsulated yellow tubercle, relation resembling that of pyæmia to focus of suppuration (Buhl, 1856). Weigert demonstrated the presence of tuberculosis of blood-vessels in a high percentage, commonest site being the *pulmonary veins* (adherent caseous glands frequently present) and thoracic duct.

TYPES OF GENERALIZED TUBERCULOSIS (Weigert).—Bacilli, without multiplying in blood, settle in organs, producing: (1) *Acute miliary tuberculosis*. (*a*) All organs affected; (*b*) Certain organs specially affected. (2) *Chronic generalized tuberculosis*. Rare. Mainly in children. Larger scattered yellow and caseous tubercles.

Acute Miliary Tuberculosis.—

GENERAL CHARACTERISTICS.—(i) Always secondary to some primary local focus; focus may be extremely small. (ii) Febrile course not exceeding a few weeks. (iii) Always fatal. (iv) Most frequent in young children, especially after measles and whooping-cough.

THREE PRINCIPAL CLINICAL TYPES. (1) *Acute general miliary tuberculosis*: symptoms 'typhoidal'. (2) *Acute miliary tuberculosis of the lungs*: marked pulmonary symptoms. (3) *Tuberculous meningitis*: marked cerebral symptoms. All intermediate forms occur. Development of pulmonary or cerebral type not uncommon in cases commencing as generalized form.

ACUTE GENERAL MILIARY TUBERCULOSIS.

(*Typhoidal Form.*)

Etiology. *Age*. Usually young: rare over 20 years.

Symptoms.—

ONSET.—Insidious progress of malaise as in enteric fever. Gradual development of feverishness, weakness, and wasting. Abrupt onset in rare instances.

PROGRESS.—Characterized by *severe toxæmia with few local symptoms*. (1) *Tongue* and skin dry. Cheeks flushed. Sweating

Tuberculosis—Acute General Miliary Symptoms, *continued.*

may occur. (2) *Pulse* rapid and feeble: rarely dicrotic. (3) *Temperature irregular*: about 103°: remittent or intermittent: inverse type not uncommon (morning rise). Rarely almost afebrile. (4) *Lungs*: often no changes, may be slight bronchitis. (5) *Spleen* often palpable. Diarrhœa unusual. (6) *Mental condition*: torpor progressing to final coma. Acute delirium rare.

TERMINATION.—Often marked pulmonary or cerebral symptoms develop (corresponding to other types): or passes through 'typhoidal' state to death in coma.

DURATION.—Usually less than a month: occasionally one to three months.

Diagnosis.—Usually extremely difficult. From:—

TYPHOID FEVER.—Often very uncertain. In tuberculosis:—

MOST DEFINITE.—(1) *Temperature irregular.* (2) No rose-red rash (3) Specific reactions: agglutination reaction and blood cultures negative. (4) Blood-count: polynuclear leucocytosis. In typhoid: leucopenia and relative lymphocytosis. (5) Fæces: no enteric bacilli. (6) Lumbar puncture: small lymphocytes may be present.

LESS DEFINITE.—Age: usually young. Spleen: less frequently palpable, but may be so in late stages. Diarrhœa: unusual, but sometimes marked: rarely 'pea-soup'. Cyanosis: bronchitis and respirations may be increased. Sweats, herpes, and petechiæ: occasionally. Signs of pulmonary tuberculosis. Choroidal tubercle: pathognomonic, but very rare.

Note.—Typhoid fever and miliary tuberculosis may co exist

SEPTICÆMIA.—Blood cultures. Septic focus.

INFECTIVE ENDOCARDITIS.—Blood cultures. Cardiac lesions

HODGKIN'S DISEASE.—Unusual types.

ACUTE MILIARY TUBERCULOSIS OF THE LUNGS.

Etiology.—Adults: previous cough or tuberculosis. Children: measles or whooping-cough, or tuberculous disease. May be no factors.

Symptoms. *Marked pulmonary symptoms.*

ONSET.—*As bronchitis*: sputum purulent, hæmoptysis rare.

ESSENTIAL SYMPTOMS.—

Cough
Dyspnœa } Severe and out of proportion to physical signs.
Cyanosis

OTHER SYMPTOMS.—Fever: 102° to 104°: may be inverse type. Rarely afebrile. Physical signs of bronchitis only.

Spleen generally palpable.

Lungs.—May be hyper-resonant. In children, often slight impairment of note and bronchial breathing at bases, from collapse.

PROGRESS AND DURATION.—*Rapid wasting and weakness.* Symptoms of cerebral type may develop. *Duration*: commonly about two weeks, usually within one to six weeks, in rare instances two months.

Diagnosis.—On essential symptoms, usually aided by etiology. Tubercle bacilli in sputum rare. Choroidal tubercles very rare.

TUBERCULOUS MENINGITIS.

(*Basal Meningitis.*)

Etiology.—*Age*: Commonest from two to five years: rare under one year. No age immune. Secondary to tuberculous focus elsewhere, often bronchial or mesenteric glands.

Morbid Anatomy.—

MENINGES AT BASE AFFECTED.—Leptomeningitis, dura mater not involved. Interpeduncular space, optic chiasma, Sylvian fissure affected: may spread over lateral surface and over pons, rarely on upper surface.

MEMBRANES.—Matted together, or purulent exudate, or milky appearance from turbid fluid in subarachnoid space over these areas, and extending along nerves.

TUBERCLES.—Size of pin's head, whitish, scanty or numerous. Situated on (a) membranes, especially in Sylvian fissure; (b) arteries (appearing as nodules), especially middle cerebral and anterior and posterior perforating arteries

LATERAL VENTRICLES.—Distended with turbid fluid, fornix and septum lucidum destroyed, and convolutions flattened (acute hydrocephalus).

CEREBRAL TISSUE under affected meninges œdematous and infiltrated with leucocytes, i.e., encephalitis present.

Occasionally: Meninges of cervical cord affected. Caseous tuberculous masses in brain substance.

Symptoms.—Described as they occur in children. Are numerous and variable.

COURSE. Prodromal period. Followed by *three stages*, duration of each about one week: (1) Stage of irritation; (2) Stage of increasing intracranial pressure; (3) Stage of paralysis or coma.

PRODROMAL PERIOD.—May follow measles, whooping-cough, or a fall. Wasting; anorexia; peevishness. Duration about two weeks, or up to six.

FIRST STAGE.—*Stage of Irritation* (of meninges and cortex). Onset often with a convulsion. Essential symptoms at onset are:—

1. Headache: intense: child puts hand to head.
2. Vomiting: cerebral type, independent of food.
3. Fever: 102° to 103°.

Other symptoms developing during this stage:—

4. Pulse: rapid at first but becomes slow and irregular (cerebral pulse).

Tuberculosis—Tuberculous Meningitis—Symptoms, ***continued.***

5. Constipation: invariable.
6. Hydrocephalic cry: short causeless scream: rarely, continuous crying.
7. Pupils contracted.

Also common: restlessness, twitchings of muscles, slight squint, photophobia, fontanelle tense; occasionally marked hyperæsthesia.

SECOND STAGE.—*Stage of Increasing Intracranial Pressure.* Irritation diminishes, viz., vomiting and headache slight. Lies on side with elbows and knees flexed. Difficulty in swallowing.

1. Drowsy but irritable: resists feeding or moving.
2. Abdomen carinated (retracted): constipation.
3. Ocular changes: (*a*) Pupils dilated or unequal, reaction to light altered; (*b*) Movements of eyes may be inco-ordinated; (*c*) Squint; (*d*) Early optic neuritis, ptosis.
4. Convulsions, or rigidity: latter may follow convulsions.
5. Temperature: lower, about 100° to 102°.
6. Pulse, slow and irregular: respiration similar, but less marked.

Head retraction not uncommon, but rarely marked.

Tache cérébrale. May be erythemata. Cheeks often flushed.

THIRD STAGE.—*Stage of Paralysis.*

1. Coma, becoming deeper.
2. Motor symptoms; (*a*) Convulsions; (*b*) Local spasms; (*c*) Paralyses; (*d*) Contractions.
3. Pupils dilated and other signs as last stage. Eyelids close partially.

Pulse rapid. Diarrhœa. Incontinence complete. May be typhoidal state. Temperature low, rising before death.

DURATION.—Three weeks common. From two to six weeks.

VARIATIONS.—(1) Acute form fatal in a few days with abrupt onset. (2) Acute form supervening on tuberculous tumour presenting symptoms of cerebral tumour.

Symptoms: Special Features.—

PULSE.—Rapid at onset, becomes slow and irregular as intracranial pressure increases (less marked under 5 years): finally rapid as heart fails.

TEMPERATURE.—High in first stage (103°), then falls (100°), may rise or be hyperpyrexial (106°) in third stage.

OCULAR CHANGES.—

PUPILS.—In first stage contracted: then dilate as intracranial pressure increases. Often unequal. On exposure to light may 'oscillate', contracting and immediately dilating. Later, dilatation increases and reaction to light is absent.

EXTERNAL MUSCLES.—(i) Squint: often early sign. (ii) Inco-ordinated movements: slow independent movements of the two eyes from side to side. Important sign, but may occur in healthy young children during sleep. (iii) Ptosis.

OPTIC NEURITIS.—Rarely intense: edge of disk blurred and vessels curved. In early stage presence usually doubtful.
CHOROIDAL TUBERCLE.—Very rare.
Conjunctival and corneal reflex lost in last stage.

MOTOR SYMPTOMS.—
CONVULSIONS.—May occur (i) At onset of first stage: solitary general convulsion; (ii) In second stage: very variable, often local spasm of one limb, etc., from cortical irritation; (iii) In third stage: may be general. Rigidity, paralyses, or contractions may follow.
PARALYSES.—Occur in second and third stages. Sometimes transient. (i) Hemiplegia: either from internal capsule or cortex (from affection of branches of middle cerebral artery); (ii) Monoplegias: various. Of cranial nerves, most often 3rd and 7th: may be syndrome of Weber.
RIGIDITY.—Invariable: often follows convulsions.
VARIOUS.—Tremors: athetoid movements: local spasms.
KERNIG'S SIGN.—Usually present. Absence of no importance.
Babinski's sign occasionally present. Knee-jerks variable; increased or diminished.

DECUBITUS.—In first two stages lies on side, elbows and knees flexed. If moved to back, returns to side. In third stage may lie on back.

Special Reactions.—
LUMBAR PUNCTURE.—Fluid under pressure. Character diagnostic, i.e., (1) Protein increased, (2) Small lymphocytes present (*see* PLEURAL FLUIDS); (3) Tubercle bacilli usually present, but often difficult to find. Fluid clear or slightly turbid.
BLOOD COUNT.—Polynuclear leucocytosis: 15,000 to 20,000.
TUBERCULIN REACTIONS.—Calmette's and von Pirquet's may be positive early, but are negative later.
TYPHOID AGGLUTINATION REACTION.—Negative: considerable agglutination not uncommon, but titre below 'positive'.

Differences in Adults. Prodromata rare. Earliest symptoms may be (1) squint (diplopia), (2) aphasia or some alteration in speech, or (3) vomiting. Less commonly (4) monoplegia or hemiplegia, sometimes with aphasia, (5) condition suggestive of hysteria. Delirium, and muscular twitchings and rigidity common, but general convulsions rare. Coma rapid and duration short (about two weeks): ascribed to unyielding adult skull.

Diagnosis.—Questions are (1) Is meningitis present? (2) If so, what is the type? Important are: (*a*) *Spinal fluid* (completely diagnostic). Agglutination reaction of serum. (*b*) Age (rare under one year: commonest 2 to 5 years). (*c*) Previous tuberculous foci.
IS MENINGITIS PRESENT?—Diagnosis from:—
TYPHOID.—Patient in relaxation, lies on back, abdomen distended. Agglutination reaction develops.
PNEUMONIA.—Especially apical. Pulmonary signs.
ACUTE GASTRITIS.—Tongue furred; no cerebral signs.

Tuberculosis—Tuberculous Meningitis - Diagnosis, *continued.*

ACUTE POLIO-ENCEPHALITIS.
OTITIS MEDIA.
In adults, from INTRACRANIAL TUMOUR, or rarely HYSTERIA.

2. TYPE OF MENINGITIS.—CEREBROSPINAL MENINGITIS is the usual type under one year: head retraction marked.

Prognosis.—Always fatal.

Treatment.—Repeated lumbar puncture eases the pain · twenty-four or forty-eight hour intervals. Careful nursing and nasal feeding in later stages prolong life.

VIII. PULMONARY TUBERCULOSIS.

(*Consumption. Phthisis*)

Classification.—Pulmonary tuberculosis occurs in following forms

ACUTE PULMONARY TUBERCULOSIS.—
1. ACUTE PNEUMONIC TUBERCULOSIS
2. ACUTE BRONCHOPNEUMONIC TUBERCULOSIS } *Acute Phthisis*
3. ACUTE MILIARY TUBERCULOSIS OF THE LUNG

CHRONIC PULMONARY TUBERCULOSIS
FIBROID PHTHISIS } *Chronic Phthisis*

ACUTE PULMONARY TUBERCULOSIS.

Acute Pneumonic Tuberculosis.

(*Tuberculous Lobar Pneumonia*)

Very rare. Usually in males.

Morbid Anatomy.—*One lobe*, usually upper, affected, or less often whole lung. *Small cavity* or caseous focus frequent, whence infection has spread probably by bronchi. *Affected area* · solid, heavy, airless, and grayish, resembling hepatization. Miliary tubercles often not obvious. May be tubercles in other lobes of same or other lung or caseous glands, thus revealing condition, even in absence of cavity or caseous focus If more chronic, may be areas of caseation or excavation: rarely whole lung caseous

Symptoms.—

ONSET.—Often typical of acute lobar pneumonia.

PROGRESS.—Symptoms and physical signs of typical pneumonia *until crisis fails to occur.* Suggestive symptoms then arising (1) Irregular temperature; (2) Rapid pulse and severe constitutional disturbance; (3) Persistence of consolidation in lungs.

SUBSEQUENT PROGRESS.—Irregular temperature, rapid wasting, sweats; prostration. Signs of cavitation develop, sputum becomes purulent

TERMINATION.—May be (1) Typhoid state and rapid death, about two weeks; (2) Gradual failure and death, about two months: usual form. In extremely rare cases, acute symptoms subside and chronic tuberculosis follows.

Diagnosis.—Rarely diagnosed from typical pneumonia until crisis fails. Differences (all of little value) may be: (1) Suspicious

family or personal history: and onset less abrupt; (2) Temperature less regular from commencement; (3) Breath-sounds faint rather than tubular: a point much emphasized.

Tubercle bacilli may be found in first week, but rarely under 10 days. Signs of cavitation may give earliest diagnosis.

Acute Bronchopneumonic Tuberculosis.

(*Tuberculous Bronchopneumonia.*)

Commonest form of 'galloping consumption' or phthisis florida, especially in children.

Morbid Anatomy.—

MACROSCOPIC.—

1. Lung studded with *grayish nodules* or, if longer duration, *small caseous masses*, ¼ to ½ inch diameter. Miliary tubercles unusual.
2. Scattered *small ragged cavities*. Large new cavities uncommon, owing to short duration.
3. Intervening areas of lung tissue show (*a*) red pneumonic consolidation, or (*b*) emphysema or œdema.
4. Old cavity or lesion not uncommon, usually at apex.
5. Bronchi contain purulent secretion.
6. Fibrinous pleurisy present.
7. *Bronchial glands* often enlarged and caseous round root of lung in children. Pneumothorax not uncommon.

Areas may be involved at different sites, *especially both apices*. In other cases, one lobe may be nearly solid, but intervening non-tuberculous portions are nearly always recognizable.

In *children*, when duration short, tuberculous nature of bronchopneumonia not always recognizable macroscopically. In slower forms caseous areas present.

HISTOLOGY. The lesion is an acute, caseating bronchopneumonia, commencing in the walls of the finer bronchioles. The nearest alveoli are affected with a catarrhal pneumonia. The tuberculous process and resulting caseation gradually extend. In small focus, the following changes are present.—

1. *Central bronchiole.*—Walls thickened and caseating. Lumen contains caseous matter.
2. *Alveoli* in immediate neighbourhood destroyed by caseation, with varying degree of fibrosis. Remnants of alveoli may be visible.
3. Surrounding zone of alveoli with thickened alveolar walls and air-spaces plugged with catarrhal products; commencing caseation present in parts.
4. Outer zone of alveoli unchanged, or with evidence of emphysema, œdema, or commencing involvement in focus.

Modes of Onset.—

ADULTS.—

(1) Abrupt onset: following overwork or strain, especially in alcoholics.

(2) Following influenza.

Tuberculosis—Acute Pulmonary—Modes of Onset, *continued.*

(3) Cough for a period: tuberculous focus, whence spread.
(4) Sequel of hæmoptysis: whence aspiration of tuberculous matter into bronchi: generally rapid progress.

CHILDREN.—Often following measles and whooping-cough.

Symptoms.—

ONSET.—Abrupt: rigors, dyspnœa, cough, high temperature, rapid pulse. Sometimes more gradual.

PROGRESS.—*Wasting and weakness marked,* often vomiting.

TERMINATIONS.—

1. Symptoms progress rapidly: hectic temperature, sweats (mainly at night), wasting, and pulmonary symptoms Typhoidal state may develop, delirium, dry tongue and skin, diarrhœa. Death in three weeks.
2. Less rapid: death about two months.
3. Improvement after some weeks and becomes chronic: rare

Physical Signs.—Early: diffuse bronchitis, both lungs. Later: areas of consolidation, especially at apex; percussion note impaired, breathing loud or tubular, râles.

Diagnosis.—

IN ADULTS.—Tubercle bacilli present early in sputum. Severity of symptoms suggestive.

IN CHILDREN.—Usually swallow sputum. Rapid wasting and weakness with bronchopneumonia suggestive.

Acute Miliary Tuberculosis of the Lungs.

(*See* ACUTE MILIARY TUBERCULOSIS, p. 128)

CHRONIC PULMONARY TUBERCULOSIS.

(*Chronic Ulcerative Phthisis.*)

Distribution of the Lesions.—

PRIMARY LESION.—*Usual Site*: In upper lobe, 1 to 1½ inch below apex, nearer posterior and external borders. Corresponding points on surface: (a) Anterior: below middle of clavicle; (b) Posterior: supraspinous fossa. Extends downwards thence—on anterior surface, about 1½ inches from sternal line. *Less common site*: Below middle and outer third of clavicle, between 1st and 2nd spaces.

SECONDARY LESIONS.—*Common sites*: (1) Lower lobe of same lung. About 1 to 1½ inch below its apex. Corresponding point on surface posteriorly: opposite 5th dorsal spine. Extends: parallel to interlobar septum, downwards and outwards. (2) Upper lobe of opposite lung. Relative frequency of these two as first secondary lesion doubtful: lower lobe probably usually earlier: almost always infected by time physical signs present at apex.

Last site to be affected: Base and anterior portion of lower lobe.

Initial lesion at base: extremely rare in adults. Less rare in children, by extension from enlarged bronchial glands.

Right apex affected somewhat more often than left.

SPREAD TO OTHER LOBES.—Caused by: (1) Inhalation of infective matter through bronchi. (2) By lymphatics from secondarily infected bronchial glands; (3) By back flow in blocked lymphatics.

CAUSE OF ORIGIN AT APEX.—*Theories*: (1) Slighter respiratory movement at apex, considered by anatomists to be minimal at exact spot where onset is most common. Results in diminished circulation in capillaries, deficient aeration, and hence weakness of tissues. (2) Connection between apical lymphatics and those of bronchial glands and lymphatic glands of neck.

Morbid Anatomy (*see* p. 137).—

The lesions are extremely variable, not only in different cases, but also in different lobes of the same specimen and in various parts of the same lobe. The general conditions are:—

1. The essential lesions of tuberculosis (*see* HISTOLOGY, p. 125) are occurring—viz., cellular changes, followed by (*a*) necrosis (and caseation), or (*b*) development of fibrous tissue ('fibrosis' or 'sclerosis'). These two sequelæ occur together, and result depends : which predominates, fibrosis tending to heal and necrosis to extend the lesion.
2. Every stage in above development may be found in small area of lung. Thus it may contain every stage of a tubercle, and also at one point fibrosis may predominate, and at another necrosis.

Summary of Usual Development of Chronic Tuberculosis.—Initial focus: wall of small or terminal bronchiole. Gray tubercle develops. Meanwhile alveoli fill with epithelioid cells. Lesion proceeds to stages of necrosis (commencing centrally), and fibrosis (peripherally). Assuming extension occurs, focus has now: (1) A central bronchiole containing mucus or, later, caseous material from alveoli; (2) The bronchiole wall and neighbouring alveoli in progressive stages of tubercle formation, necrosis and caseation, and some degree of fibrosis. (3) A surrounding zone of alveoli showing catarrh, as in bronchopneumonia. (4) External to this: (*a*) patches of collapse of alveoli and of 'emphysema' (more correctly, alveolar distention); (*b*) miliary tubercles, spreading from initial focus by lymphatics.

Lesions as affecting Various Tissues of Lung.—

SMALL BRONCHI AND BRONCHIOLES.—Chronic tuberculosis usually commences in wall: elementary gray tubercle forms, as described in GENERAL HISTOLOGY, forming peribronchial tubercle.

ALVEOLI AND ALVEOLAR WALLS.—(1) *Alveoli*: In lobule of affected bronchiole, alveoli fill with epithelioid cells and a varying number of leucocytes, generally scanty. Necrosis of these cells occurs, thus forming a plug within the alveolar walls. (2) *Alveolar Walls*: Early change is cellular infiltration and some thickening of fibrous tissue. Necrosis is, in general, later in walls than in alveolar contents: on occurrence it forms a fused caseous mass with alveolar contents and uniting with the peribronchial tubercle. Thus at this stage from lumen outwards are seen: (*a*) *Caseous area*.

Tuberculosis—Chronic Pulmonary—Morbid Anatomy, *continued.*

consisting of original tubercle fused with the caseated alveolar contents and alveolar walls. May be occasional giant cell. (ii) Area in which alveolar walls are thickened but still present, with caseous contents of air-space. (iii) Area in which alveolar walls show early changes and contents are epithelioid cells.

Subsequent progress (and even extension to this stage) depends on predominance of one or other of the two tuberculous changes: (1) Necrosis: Caseous mass formed. May rupture into bronchus, forming small cavity. (2) Fibrosis: Growth of connective tissue from wall of bronchus, alveoli, or interlobular septum may arrest growth at any point, enclosing mass in fibrous capsule.

EXTENSION OF TUBERCULOSIS.—Occurs by: (a) *Miliary tubercles* in neighbourhood of primary focus: bacilli carried by lymph. Fusion follows with parent lesion. (b) Aspirated matter infecting neighbouring bronchioles, or even more distant sites and other lobes.

ARTERIOLES AND CAPILLARIES. Destroyed by tuberculous progress. *No vessels ever present in tubercles.* Rupture of capillaries causes the slight early hæmoptysis (*see also* CAVITIES).

FIBROUS TISSUE.—*Arrest of Tuberculosis.* All fibrous tissue within tuberculous zone tends to proliferate, amount varying with rapidity of spread. May arrest progress and cause 'healing'.

1. In alveolar walls and small bronchioles. May result in: (a) Subsequent degeneration to a granular débris, uniting with any caseous material; (b) Permanent fibrous tissue and arrest of progress. Not common.
2. In interlobular septa. Similar, but more often permanent. May organize later, with development of new blood vessels and, contracting, assist in formation of 'fibroid lung'.

RESULTS OF FIBROUS CAPSULATION and the healing of a focus:—

1. A 'puckered scar': fibrous tissue contracts and tuberculous process arrested. Frequent at apex without other signs of tuberculosis, as remnant of former lesion.
2. Caseous nodule with thick fibrous capsule: central matter still infectious, and rupture may cause acute tuberculosis.
3. Calcification of nodule arising as above, due to subsequent impregnation with lime salts: not infectious: very hard, may become loose and be expectorated as 'lung stone'.

In walls of cavities, fibrous-tissue formation results in slowing or, more rarely, arrest of advance.

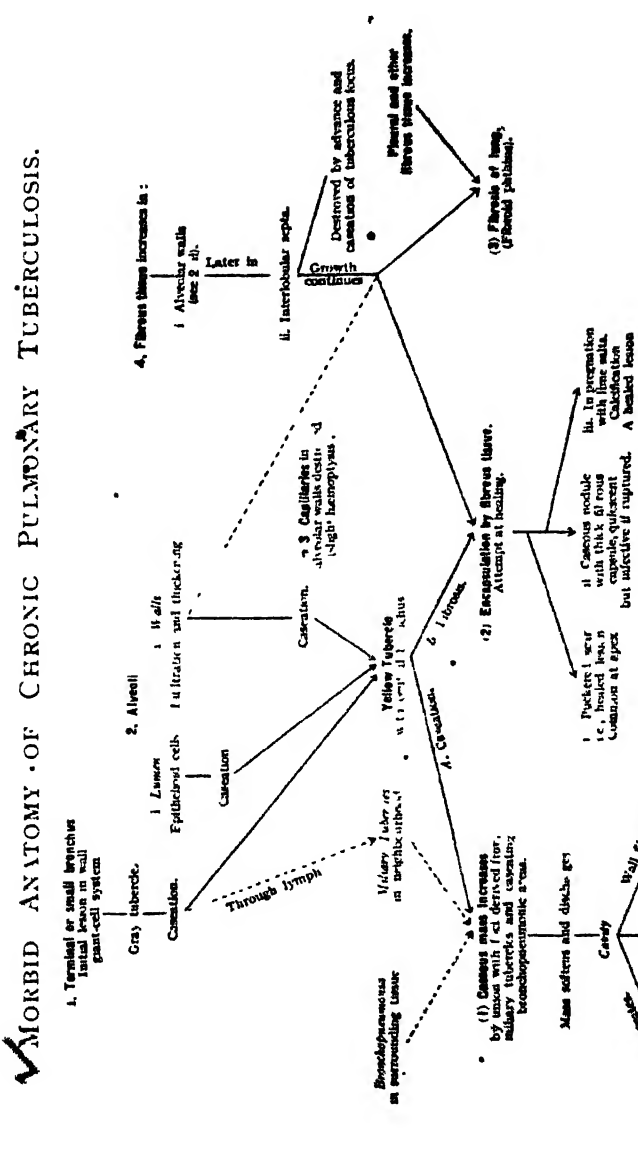
LUNG TISSUE OUTSIDE DEFINITE NODULE.—May show:—

(1) Catarrhal pneumonia. As in ordinary bronchopneumonia. Accounts for wide area of consolidation in early phthisis. May involve larger portion of lobe. Probably of tuberculous origin. Macroscopic appearance may be: (i) Resembling red hepatization; (ii) Homogeneous and gelatinous, *infiltration tuberculeuse* (Laennec); (iii) Numerous opaque points from degeneration of alveolar contents.

(2) Patches of alveolar collapse: from blockage of bronchioles.

(3) Patches of 'emphysema'—or (more correctly) distention of alveoli.

MORBID ANATOMY OF CHRONIC PULMONARY TUBERCULOSIS.



Tuberculosis—Chronic Pulmonary—Morbid Anatomy, *continued.*

CAVITIES.—Caseous matter may soften by ingress of fluid, and then discharge through ulcerated bronchus, thus forming a vomica or *cavity*. Size varies from small pea to, rarely, whole lobe. *Of following types*, but all may co-exist:—

FRESH ULCERATIVE CAVITY.—Soft ragged wall, no limiting membrane. In acute phthisis often numerous and small. Also in chronic forms where extension is taking place.

FIBROUS CAVITY.—Wall definitely fibrous but discharges pus. Contents resemble nummular sputum. Strands of blood-vessels or bronchi may persist. Extends slowly. Constitutes largest form.

QUIESCENT CAVITY.—Smooth fibrous-tissue wall. Usually small. Is maximum healing of a cavity.

Fibrous tissue near cavity tends to increase and adjacent pleura thickens. *Pleural thickening* common at apex with one or more quiescent cavities.

BLOOD-VESSELS.—Obliterated by inflammation, but are last tissue affected: thus often persist as strands running through cavities, with blood circulating. May rupture from (a) erosion of wall, or (b) formation of aneurysms: serious hæmorrhage results.

PLEURA.—1. Dry.—Always affected in chronic phthisis. May be:—
2. Dry pleurisy—thin adhesions.
3. Caseous pleurisy—great thickening of pleura.
4. Effusions: clear, hæmorrhagic, or purulent: usually sterile, occasionally pneumococci or pyogenic cocci.
5. Pneumothorax: from rupture of caseous nodule.

BRONCHI.—Inflammation spreads up from small bronchi. Aided by coughing, may result in bronchiectasis. In larger tubes, chronic catarrh.

BRONCHIAL GLANDS.—In acute phthisis, enlarged and œdematous: miliary tubercles and caseous foci. In chronic phthisis: either caseous, hard and calcified, or softened.

PIGMENTATION.—When chronic, some pigmentation of fibrous tissue almost invariable, old lesions being of slaty colour from carbon particles.

Other Organs Affected.—*Tuberculosis* may be present in (arranged in order of frequency): (1) Lymphatic glands, (2) intestines, (3) larynx, (4) spleen. Less common: kidney, brain, liver; pericardium rare, and endocardium very rare.

Stages of Phthisis.—

TURBAN'S CLASSIFICATION.—*First stage*: Physical signs present in one lobe. *Second stage*: Physical signs present in two or more lobes, usually three. *Third stage*: Physical signs of widespread disease or cavities. 'Open tuberculosis' is applied to cases with tubercle bacilli in sputum, and 'closed' to those without tubercle bacilli. In the former, tuberculous matter must be in communication with a bronchus.

Three clinical stages formerly described supposed to correspond to (1) Growth of tubercles; (2) Caseation; (3) Excavation. Nowadays these are not accepted as accurate or of value.

Symptoms.—

MODES OF ONSET.—

1. LATENT.—Lesions advance far without symptoms.
2. BRONCHITIS, or a 'neglected cold on the chest'. Common.
3. DYSPEPSIA.—Vomiting.
4. ANÆMIA.—Simulates chlorosis in girls.
5. ENLARGED CERVICAL OR AXILLARY GLANDS.—May precede pulmonary symptoms for years.
6. HÆMOPTYSIS.—This may be followed by (i) Rapid phthisis, from aspiration of infective matter; (ii) Slow development.
7. PLEURISY.—(i) With effusion: signs may be present after absorption or develop later. (ii) Dry: e.g., friction at apex.
8. LARYNGEAL SYMPTOMS.—Hoarseness and irritability of throat. Tuberculous laryngitis almost always secondary to lung, but may cause first symptoms.
9. Rarely, with attacks resembling ASTHMA or MALARIA.

Note.—*Acute* pulmonary tuberculosis commences with symptoms of (1) Pneumonia or bronchopneumonia; (2) Severe general infection.

SUMMARY OF CHARACTERISTIC GROUP OF SYMPTOMS.—(1) Cough, (2) Sputum (hæmoptysis); (3) Loss of appetite; (4) Loss of weight, (5) Sweating, especially at night. Also (6) Fever; (7) Rapid pulse; (8) Clubbing of fingers.

CLASSIFICATION OF SYMPTOMS.—

LOCAL.—(1) Cough; (2) Sputum; (3) Hæmoptysis. Less frequent are (4) Pain; and (5) Dyspnœa.

GENERAL OR CONSTITUTIONAL.—(1) Fever; (2) Pulse rapid; (3) Sweating; (4) Wasting; (5) Loss of appetite. Less definite are: (6) Facial appearance; (7) Clubbing of fingers; (8) Anæmia.

Local Symptoms.—

COUGH.—*Most frequent early symptom*, usually persists throughout. Absence extremely rare. Nothing characteristic. Most at night and early morning. Worst in rapid advance, and disease of larynx and trachea, but no constant relation to severity of lesion. May cause vomiting, especially in the paroxysms. Food may cause attack. *Early stages*: often dry and hacking. *Later*: looser, with sputum. *With cavitation*: often paroxysmal, especially morning. *With laryngeal tuberculosis*: husky and ineffective.

SPUTUM.—*Rarely absent*, but patient may swallow sputum until instructed to expectorate. Not characteristic until late stages when nummular. Important data are: presence of (*a*) tubercle bacilli, (*b*) blood.

Tuberculosis—Chronic Pulmonary—Local Symptoms, *continued.*

CHARACTER.—Varies with stage:—

Early: mucoid, from degenerated epithelial cells.

Later: greenish purulent masses, very suggestive of phthisis.

Cavities present: 'nummular', solid airless masses sinking in water.

AMOUNT.—In rapid cases with much cough may be 500 c.c. daily: with cavities, most in morning.

ODOUR.—Sweetish. Fœtid only with complications, e.g., bronchiectasis, gangrene.

BLOOD.—May be present from hæmoptysis.

MICROSCOPIC EXAMINATION.—(i) Tubercle bacilli. (ii) Elastic tissue: proof of destruction of tissue. Nowadays of little importance. Boil with equal amount of 10 per cent caustic soda, dilute with water, and examine deposit.

HÆMOPTYSIS.—Occurs in 60 to 80 per cent of cases, and is seen at two stages:—

EARLY STAGE.—Amount small (half ounce): from eroded capillaries. Never fatal, but often early symptom.

LATER STAGES. From cavities: may be profuse. Source (1) Aneurysm on vessels, e.g., pulmonary arteries, size from pea to orange; (2) Rupture of vessel persisting in cavity, less often. *Occasionally, though rarely, fatal.*

MODE OF OCCURRENCE:—

Onset usually sudden, salt taste in mouth; may follow mental excitement or exertion. Patient aware of origin from lungs. Causes great mental alarm and depression.

Characters: red, frothy, alkaline. Occasionally swallowed and, later, vomited.

Sputum tinged for several days subsequently.

Recurs usually several times.

Sequelæ may be: (1) Rise of temperature few days later; (2) *Rapid progress of phthisis* (spread by aspiration of blood into other bronchi)

RELATION OF HÆMOPTYSIS TO TUBERCULOSIS.—True hæmoptysis is almost invariably of tuberculous origin, and should be so treated. Three groups recognizable when occurring in persons previously considered healthy:—

1. Physical signs and tubercle bacilli already present. Inquiry reveals previous ill-health.
2. No physical signs or tubercle bacilli, but these appear shortly after.
3. No subsequent ill-health or symptoms (about 15 per cent). Probably all are of tuberculous origin.

When following trauma to chest or severe exertion, groups are similar, about half becoming tuberculous.

PAIN.—Some pain not uncommon, but usually slight:—

From pleurisy: usually felt over lower thorax, occasionally apex or scapula.

Vague pains, probably muscular from coughing.

DYSPNŒA.—

Infrequent in uncomplicated chronic tuberculosis: absence ascribed to acquired tolerance during the slow progress: respiration may be increased in rapidity.

Occurs with complications: (1) Outbreak of acute miliary tubercles; (2) Bilateral bronchopneumonia, or with emphysema; (3) Pneumothorax; (4) Cardiac failure, as in fibrosis of lung.

General or Constitutional Symptoms.—

FEVER.—Early and extremely important sign, and the *most valuable measure of severity and progress of the disease.* Due to absorption of toxins, i.e., auto-inoculation, resembling tuberculin injection.

Records must include afternoon, not later than 6 p.m., when usual maximum. Rectal temperature necessary for all cases under treatment: exercise may cause no rise in mouth.

Normal healthy range in rectum: maximum in afternoon, at rest, 98·4°; difference from mouth temperature varies with individual, but remains fairly constant, average 1° higher (range about 0·6° to 1·8°)

Early stages of phthisis: temperature continuous or remittent, range varies with severity. Effect of rest of great importance; rapid fall favourable. With rest in bed, even *an occasional temperature* of 99° in the mouth (e.g., three in 14 days) is a sign of activity of disease, and of great diagnostic importance in early doubtful cases.

Inverse type (higher in the morning) not uncommon.

Later stages, caseation and cavity formation: *intermittent* 'hectic' temperature. Rises to 104°. Maximum at 6 p.m. Falls to normal in morning with sweating.

Effect of exercise: When temperature at rest is normal, after gentle exercise rectal temperature may be 101° (even in healthy persons); should fall to normal in half hour. *With active disease* may persist 2 to 3 hours, from auto-inoculation. *Rise continuing after cessation of exercise* is sign of excessive auto-inoculation.

PULSE.—Rate increased: may persist when temperature normal if disease active, and hence of importance. With active phthisis is rarely below 84.

SWEATING.—Often drenching, especially *during night and early morning*. Sometimes early symptom. In later stages very distressing.

WASTING.—Pronounced, whence name of disease. Commences early, often before other signs. Weight is important index of disease and needs careful record. Loss of strength also present.

LOSS OF APPETITE.—Usually early: especially for fat. Extreme nausea and vomiting not uncommon.

FACIAL APPEARANCE.—Pallor common, with faded yellow complexion. In later stages 'hectic' flush occasionally. Rarely pigmentation

Tuberculosis—Chronic Pulmonary—General Symptoms, *continued.*

CLUBBING OF FINGERS.—Important from easy recognition, but rare in early stages. Pulmonary osteo-arthropathy rare.

ANÆMIA is common but not constant in early stages: when present the colour index is low. The leucocytes are normal, or diminished in number.

Physical Signs.—

Normal differences at apices.—At right apex, breath-sounds usually louder, expiration more prolonged, vocal resonance louder and sometimes tactile fremitus more marked than on left. Attributed to right bronchus being at higher level than left.

Summary of Progress of Important Signs.—

EARLY SIGNS.—Condition is *apical* bronchitis with surrounding small areas of consolidation (*Turban's First Stage*). (1) *Fine crepitations at apex*, localized and persistent, and not removed by coughing. Commonest first sign. Other early and sometimes initial signs: (2) Slight delay or deficiency of expansion and flattening at apex (less frequently initial sign); (3) Percussion note slightly impaired; (4) Breath-sounds diminished, or, less often, harsh, with prolonged expiration.

LESION PROGRESSING, but still early.—Consolidation increasing; other lobes also showing early signs (*Turban's Second Stage*). (1) Deficient expansion and flattening; (2) Impaired note; (3) Crepitations; (4) Breath-sounds more definitely harsh and expiration prolonged; also (5) Whispering pectoriloquy and bronchophony. Early signs usually commencing to make their appearance at other sites.

LESION WELL DEVELOPED AT APEX. Caseation, softening, pleura affected (*Turban's Second* or *Third Stage*).

INSPECTION AND PALPATION.—Clavicle prominent, flattening of apex, deficient expansion.

PERCUSSION.—Impaired note.

AUSCULTATION.—Breath-sounds more tubular. Râles louder and larger. Whispering pectoriloquy and bronchophony. Signs usually progressing at other sites.

CAVITY.—*Auscultation* important: 'post-tussic suction' especially valuable. Breath-sounds bronchial, and râles develop, and voice-sounds loud and altered in tone.

Note.—For Turban's classification, *see* STAGES OF PHTHISIS (p. 137).

Physical Signs according to Methods of Examination.—

INSPECTION.

1. ALTERATIONS IN EXPANSION AT AFFECTED APEX.—(i) Delayed movement, often very early; (ii) Deficient expansion, also may be early.
2. FLATTENING AT APEX.—From muscular wasting, fibrotic contraction, and pleuritic adhesion. As early sign, rare. May remain from healed lesion, with other signs slight or absent.

3. CLAVICLE PROMINENT.

Other changes may be (not early) :—

Wasting of shoulder-girdle muscles. Slight scoliosis.

Cardiac pulsation increased in left apex affection.

Diminished expansion of affected side, shown by measurement.

NOTES ON INSPECTION.—

Phthisis frequent with any form of chest, but two special types described : (i) 'Alar' or 'pterygoid' chest, long and narrow, costal angle acute, ribs dropped, scapulæ 'winged'; (ii) 'Flat' chest, anteroposterior diameter small. Sternum often depressed and costal cartilages prominent.

Inspection, and also some percussion, signs may be accentuated by deep breathing, but auscultation during quiet respiration must precede this.

Inspection and palpation may suggest but never diagnose early stage, but in 'fibroid lung' form the most valuable signs.

Expansion of apices best tested from behind by looking over shoulders, also by placing fingers over clavicles with thumbs meeting at spines of vertebræ. When examining from in front, place hand flat over apices.

PALPATION.—Confirms inspection.

VOCAL FREMITUS.—*Increased throughout disease* except with much pleural thickening or with effusion.

PERCUSSION.—

EARLY STAGE. —*Slightly impaired note.* Often present on first examination, confirming râles. Earliest over clavicle, middle and inner third, and just above and below : posteriorly, in suprascapular fossa (upper lobe) and interspinous area (lower lobe, usually in 'second stage').

CONSOLIDATION INCREASES.—Dullness becomes more definite.

CASEATION AND EXCAVATION.—As cavity forms, dullness may diminish (*see* p. 145).

VARIATIONS AND SPECIAL DIFFICULTIES.—In early stage, note may be within normal limits though foci and crepitations are present, owing to intervening lung tissue. Lung may be emphysematous, giving more resonant note than normal.

With small cavities at apex, note may be normal but with definite auscultatory changes, or may be hyper-resonant.

Impaired note with feeble breath-sounds results from pleural thickening and some consolidation.

NOTES ON PERCUSSION.—

Light percussion reveals the slighter changes.

Compare sides in same phase of respiration : test in full inspiration, also in full expiration if doubtful.

Apex above clavicle : percuss from behind, recording as finger-breadths of resonance, normally 3 on right, and 2 to 3 on left.

Myotatic irritability common in advanced cases : of no diagnostic value.

Tuberculosis—Chronic Pulmonary—Physical Signs, *continued.*

Krönig's Apical Resonance Areas (practical value not fully established).—Normal band of resonance passes from above clavicle over shoulder, mapped by very light percussion. Width two inches at narrowest part. In phthisis: (*a*) Isthmus becomes narrower; (*b*) Margins blurred, especially inner.

AUSCULTATION.—

1. BREATH-SOUNDS.—

Earliest changes.—(*a*) Feeble, especially inspiration, with expiration prolonged: bronchial inflammation results in lobular collapse and lessened air entry. (*b*) *Harsh with expiration prolonged*: due to consolidation. Either may precede crepitations and dullness, (*a*) more frequently. 'Cog-wheel' rhythm frequent, but also in nervous people: not diagnostic: due to interrupted respiration.

Later.—Inspiration harsh, expiration prolonged.

Consolidation.—Bronchial or tubular breathing. (Râles)

Cavity.—Loud breath-sounds (*see* p. 145).

Unaffected portions of lung.—Harsh or puerile.

Breath-sounds may remain feeble, and onset of tubular breathing be delayed owing to obstruction of bronchi with resulting areas of collapse, and to irregularity of consolidation.

2. ADVENTITIOUS SOUNDS.

Early change.—*Persistent fine crepitations* at apex on inspiration. Most frequent first sign. Termed 'subcrepitant' since, coming from bronchi, they are less fine than Laennec's 'crepitant' râles of pneumonia. *Value of râles needs great attention*: auscultate on (1) quiet respiration; (2) deep inspiration; (3) 'cough and deep breath'. Characteristic are: *crepitations localized to one area, persistent on repetition, and not removed by coughing*, i.e., proof of apical bronchitis. *Note.*—(*a*) Negligible: Crepitations on first deep breath, disappearing on repetition. (*b*) Tuberculous: (i) Persistent in one area with quiet breathing and deep inspiration, (ii) At end of inspiration following cough, and present on repetition, though absent with quiet breathing: caused by forcible inspiration necessary to open obstructed bronchi.

Caseation and softening.—Râles louder and bubbling, i.e., moist sounds. (Percussion note impaired.)

Cavity.—Râles loud and resonant, especially on coughing, may be metallic or amphoric. Absent if cavity dry (rare).

3. VOICE-SOUNDS.—Increased throughout disease.

Whispering pectoriloquy and bronchophony.—Especially above clavicles; early suggestive signs, due to consolidation.

Cavity.—Above greatly exaggerated (*see* p. 145).

OTHER AUSCULTATORY PHENOMENA.

PLEURAL RUB.—May be early at apex, or at any stage.

CARDIORESPIRATORY SYSTOLIC MURMUR Due to heart expelling air from lung tissue, occurs in early tubercle or in large cavities, and also in *normal thin or nervous people*. Best heard anteriorly during inspiration

Lappet of lung over heart may cause (i) If consolidation, *clicks* synchronous with heart beat, due to compression by heart, (ii) Pleuro pericardial rub

INCREASED CONDUCTION OF HEART-SOUNDS toward diseased apex

SYSTOLIC MURMUR IN SUBCLAVIAN ARTERY —Ascribed to pressure by thickened pleura.

Physical Signs of Cavity.—

PERCUSSION —*Note altered*, either impaired (or even dull), or tympanitic if cavity large Occasionally (1) Practically normal, if pleural thickening and consolidation are slight, (2) 'Cracked-pot' sound large cavity auscultated while mouth open (uncommon), (3) Amphoric very large cavities, (4) Wintrich's sign, note varies with mouth open or closed (little value)

AUSCULTATION —

BREATH SOUNDS —Altered 'blowing' or tubular, cavernous or definitely amphoric

ADVENTITIOUS SOUNDS —Coarse, loud, ringing râles especially on coughing, May be metallic tone 'Bell sound' very rare Rarely cavity dry, and râles absent

VOICE SOUNDS —Vocal resonance, especially cough, and whispering pectoriloquy exaggerated greatly

POST-TUSSIC SUCTION —On drawing a deep breath after a cough, a hiss is sometimes audible as air enters cavity through narrow orifice Most valuable sign when present, distinguishing cavity from consolidation

NOTES ON CAVITY —(i) May be no characteristic sign, even with large cavity (ii) Diagnosis depends on auscultation, especially post tussic suction, (iii) Consolidation near large bronchus causes closely similar signs (pseudocavernous), viz, high pitched percussion note tubular breathing sonant râles Distinction between tubular and cavernous breath sounds is slight, latter more intense

FIBROID PHTHISIS.*

(*Fibroid Lung Fibrosis of the Lung*)

Commonly the slowly developing sequel of chronic tuberculosis, commencing as (1) Ordinary chronic tuberculous bronchopneumonia, (2) Chronic tuberculous pleurisy Onset and progress insidious, and very chronic

Symptoms. Slight chronic for ten or twenty years (i) *Cough*, often paroxysmal (ii) *Dyspnœa on exertion*. (iii) *Sputum* purulent may be fœtid

* *See also* CHRONIC INTERSTITIAL PNEUMONIA ('Cirrhosis of the Lung.')

Tuberculosis—Chronic Pulmonary—Fibroid Phthisis, *continued.*

Physical Signs on Affected Side.—Very characteristic: *Diagnosis mainly by inspection and palpation.* Little difference between non-tuberculous and tuberculous forms, but in latter case *cavities common at apex, and often changes in opposite lung.*

INSPECTION, MEASUREMENT, AND PALPATION.—Affected side diminished in volume: often markedly. Chest sunken. Shoulder lower. Expansion slight. Apex beat greatly displaced. Heart impulse often increased (especially left lung). Tactile vocal fremitus diminished, except with cavities.

PERCUSSION.—Impaired, but dullness rarely marked, varies with cavitation and thickness of pleura. Cardiac dullness displaced.

AUSCULTATION.—Breath-sounds usually feeble and bronchial, but vary with cavitation. Adventitious sounds vary with cavitation and bronchiectasis. Cardiac murmurs common (partly from displacement of heart).

Complications.—(i) *Bronchiectasis very common*; (ii) Hypertrophy of heart, especially right ventricle; (iii) Hæmoptysis; (iv) Emphysema in opposite lung—may mask foci of tubercle; (v) Repeated attacks of bronchitis.

Terminations.—(1) *Cardiac failure*; (2) Extension of tuberculosis; (3) Profuse hæmoptysis; (4) Amyloid disease, rarely.

VARIOUS FORMS OF PULMONARY TUBERCULOSIS.

Emphysematous Form.—Emphysema common in non-tuberculous portions of an affected lung or in opposite lung, and may mask tuberculous foci when developing. In some cases history and physical signs of chronic phthisis may be those of emphysema and bronchitis. Diagnosis of tubercle difficult; *suggested* by wasting, thorax flat instead of round, occasionally areas of dullness, and also by hæmoptysis, and *proved by presence of tubercle bacilli.*

Pleuritic Form.—*Tuberculosis frequently commences* with pleurisy; may be dry or with effusion, onset insidious or acute. Thickened pleura alone may remain. May be recurrent.

In Old Age.—Usually latent, with slow course. Often masked by emphysema and chronic bronchitis. Revealed by tubercle bacilli.

In Infancy.—Chronic tuberculosis unusual. Acute tuberculosis more frequent than in adults.

Peribronchial or Hilus Tuberculosis.—In children, pulmonary tuberculosis may arise by extension from tuberculous glands around the bronchi, constituting *peribronchial* or *hilus tuberculosis.* The earliest physical signs are occasionally at the base. In adults, hilus tuberculosis is very rare.

Basal Phthisis.—Signs commencing at base. Extremely rare in adults; usually unrecognized foci at apex.

COMPLICATIONS OF CHRONIC PULMONARY TUBERCULOSIS.

Respiratory System.*—

LARYNX.—Often affected. Important from distressing later symptoms. Due to direct inoculation from sputum.

FREQUENCY.—At autopsy about 50 per cent; during life symptoms in about 20 per cent.

SYMPTOMS.—Early, huskiness. Later, extreme dysphagia; also aphonia or ineffectual cough (*see* TUBERCULOUS LARYNGITIS).

EMPHYSEMA.—Common and may mask foci. Frequent in the unaffected (or less affected) lung.

PLEURA.—Invariably affected, but adhesions often form without symptoms. Symptoms occur from:—

1. Dry pleurisy: Very common in early stages; local or extensive.
2. Pleurisy with effusion: More common at onset than during course, but may be recurrent. During course is rarely hæmorrhagic.
3. Purulent effusion: Rare except with pneumothorax. Unfavourable, as pus cannot be absorbed and resection of rib does badly.

BRONCHIECTASIS.—Common in fibroid phthisis.

PNEUMOTHORAX.—Common. Mortality high. Three groups: (1) Fatal in few hours; (2) Fatal in few weeks (fluid develops); (3) Beneficial, rare. (*See* PNEUMOTHORAX.)

GANGRENE.—Rare. Usually confined to walls of a cavity.

GLANDS.—Bronchial, mediastinal, and tracheal glands often affected (*see p.* 158).

PNEUMONIA.—Bronchopneumonia common and often serious. True lobar pneumonia rare. Tuberculous exacerbations may simulate pneumonia.

Cardiovascular System.—

HEART.—Often small. Impulse may be exposed by retraction of lung, especially left apex. Pulmonary systolic murmurs frequent.

ENDOCARDITIS.—Systolic murmur at mitral area not uncommon; valves may be unaffected, but endocarditis is not infrequent. Tuberculous endocarditis is extremely rare.

PERICARDITIS.—Very rare.

Alimentary Canal.—

TONGUE.—Occasionally shallow tuberculous ulcers, direct infection by sputum.

ŒSOPHAGUS AND STOMACH.—Infections of great rarity.

ANOREXIA.—Very early symptom; especially for fats; may be extreme. *Nausea and vomiting*, sometimes early, very frequent in later stages; may follow cough. Cause of symptoms not known, possibly vagal stimulation.

* For further details, *see* TUBERCULOUS LARYNGITIS, PNEUMOTHORAX, etc.

Tuberculosis—Chronic Pulmonary—Complications, *continued.*

INTESTINE.—Diarrhœa is a frequent late symptom. May be due to: (1) Intestinal catarrh—main cause. (2) Tuberculous ulceration, usually in last few feet of ileum, but may be anywhere: most frequent site of secondary infection (in 75 per cent of post-mortems). Rarely perforates. (3) Amyloid disease.

TUBERCULOUS PERITONITIS.—Rare in phthisis.

FISTULA IN ANO.—Common: tuberculous origin.

Nervous System.—*Complications uncommon.* Include: coarse tuberculous masses, most frequently in cerebellum; tuberculous meningitis. Hopefulness traditionally present in last stages ('spes phthisica').

Genito-urinary System.—Genito-urinary tuberculosis uncommon in chronic phthisis. Albuminuria may be: (1) Febrile; (2) Amyloid disease; (3) Rarely nephritis.

Blood.—Secondary anæmia develops, but is not usually an early symptom. Leucopenia in early stages; polynuclear leucocytosis in later stages.

Bones and Joints.—Secondary disease uncommon. Chronic arthritis not infrequent, ascribed to lowered resistance.

Cutaneous System.—Pigmentation occurs occasionally: less frequent than with peritoneal tuberculosis. Pityriasis versicolor common. Clubbing of fingers common.

Amyloid Disease.—(1) Kidney: polyuria, albuminuria, casts. (2) Intestine: diarrhœa. (3) Spleen and liver: enlarged.

Concurrent or Secondary Infections.—Various bacteria especially *pneumococci, streptococci,* and *Micrococcus catarrhalis,* present in sputum. May cause bronchitis and fever, and such conditions may improve with vaccines, but general relationship still doubtful.

In examining sputum, wash mouth with sterile water, collect sputum in sterile vessel, and take cultures from centre of masses, to avoid oral bacteria.

DIAGNOSIS OF CHRONIC PULMONARY TUBERCULOSIS.

The difficulty occurs in the early cases. Family history of tuberculosis, previous illness or worry, phthinoid chest must be considered, but affect prognosis rather than diagnosis.

Diagnosis rests on: (1) Symptoms; (2) Physical signs; (3) Sputum (the presence of tubercle bacilli is obviously conclusive); (4) Specific reactions; (5) X rays.

Any one of the first three may be sufficient for a positive diagnosis. Neither specific reactions nor X rays justify a definite diagnosis if contra-indicated or unsupported by the previous factors.

Symptoms.—Of greatest importance in diagnosis are :—

LOSS OF WEIGHT, of strength, and of appetite; anæmia, unaccountable and progressive. *Weight* to be recorded weekly and loss noted.

COUGH in the young, persistent, and worse in morning, is suspicious.

HÆMOPTYSIS.—Almost always tuberculous.

NIGHT SWEATS.

FEVER.—In doubtful cases take temperature two-hourly for ten days, and note effect of exercise on rectal temperature.

PULSE.—Rapid.

Physical Signs.—Earliest are: changes in breath-sounds, crepitations, and slightly impaired resonance at the apex. Slight signs need repeated examinations, and must be accepted with special caution in absence of symptoms.

Sputum.—Absence of tubercle bacilli does not negative diagnosis based on sufficient symptoms and signs. In doubtful cases make three examinations, and employ Ellermann and Erlandsen's or some similar method.

Specific Reactions.—Tubercle bacilli produce no soluble toxins, but a focus in the body results in changes recognizable at a distance from its site. Such changes are employed for diagnosis, and, in some cases, also for treatment. Unfortunately these changes (1) may remain after lesion has healed; (2) may occur with infections too small to constitute clinical 'disease'; and (3) are not always present in cases with 'disease'.

Methods in use are of two types :—

(1) PHENOMENA OF HYPERSENSITIVENESS.—(i) Tuberculin reaction; (ii) Calmette's ophthalmic reaction; (iii) Von Pirquet's cutaneous reaction.

(2) PHENOMENA OF IMMUNITY.—(i) Tuberculo-opsonic index; (ii) Complement-fixation test; (iii) Agglutination.

PHENOMENA OF HYPERSENSITIVENESS.—Koch found that when an animal with a subcutaneous focus of tubercul. s received an injection of tubercle bacilli, the focus necrosed and h aled. On this was based the original tuberculin treatment. Koch believed that the toxins injected, added to the amount present in the focus, produced an increase of necrosis, with subsequent separation of the focus. It is now recognized that the phenomenon depends on hypersensitiveness or anaphylaxis such as follows injections of egg-albumen or most foreign proteins. The following reactions are tests of hypersensitiveness, a positive result proving that the organism has previously received an inoculation with a similar toxin, but not necessarily proving anything as to amount, presence, or date of occurrence.

ORIGINAL TUBERCULIN REACTION.—Subcutaneous injection of 0·25 c.c. 'old tuberculin' in healthy persons produces slight malaise, fever, and tendency to cough, commencing in four to six hours and passing in twenty four hours. But injection

Tuberculosis -Chronic Pulmonary -Diagnosis, *continued.*

of 0·01 c.c. in infected persons produces: (1) More severe general symptoms; (2) Reaction at tuberculous focus, well seen in lupus; (3) Local reaction at site of injection.

Method.—Subcutaneous injection of ·001 c.c. 'old tuberculin'. Temperature taken three-hourly for three days previously. Rise commences in about six hours, and is maximum in twelve to twenty-four hours: injection convenient in early morning. Rise of 1° is positive. If no rise, inject ·002 c.c. two to three days later. If no rise, inject ·005 c.c. two to three days later. If third injection is negative, it is considerable evidence against active focus, but a positive result here is vitiated by effects of earlier doses.

In case of a rise of 0·5°, repeat same dose two to three days later: if doubtful rise repeated, abandon test.

Contra-indications to test.—Fever. Recent hæmoptysis.

CALMETTE'S OPHTHALMIC REACTION.*—One drop of special tuberculin is placed in conjunctival sac. For children, half dose. If positive, *injection of conjunctiva* results: maximum about eight hours, passing off in twenty-four to thirty-six hours (but reaction and recovery may be longer).

Contra-indication.—Conjunctival disease. Apart from this, risk is very slight.

VON PIRQUET'S CUTANEOUS REACTION.—On the carefully cleansed skin of the flexor surface of the forearm are placed two drops of 'old tuberculin' about 4 inches apart with a drop of normal saline (as control) between them. The skin is scarified through the drops without drawing blood: small pieces of cotton wool are placed on the drops and removed after ten minutes. After subsidence of initial redness, if test is positive, inflammation occurs round the tuberculin spots, and a papule of ½-inch diameter forms in twenty-four hours, with maximum in forty-eight hours.

The last two tests are frequently positive after twelve years of age even in absence of clinical disease, and are negative in moribund cases. A positive reaction is of little value after childhood.

PHENOMENA OF IMMUNITY.—

TUBERCULO-OPSONIC INDEX.—In normal persons, with or without exercise, the 'opsonic index' lies within normal limits on repeated examinations. Allowing for experimental error, 0·8 to 1·2 is normal. In localized tuberculosis, index is usually 0·3 to 0·8. In tuberculosis with general disturbance, a single test of the index may be high, normal, or low: *the important feature is variation on repetition*, ascribed by Wright, and generally accepted, as due to auto-inoculation. Artificial auto-inoculation: the index may be measured before and after moderate exercise, usually a walk, variation indicating auto-inoculation (Inman).

The opsonic index has not produced convincing results in practice, and has fallen into general disuse.

* Priority for the suggestion of using the conjunctiva belongs to Wolff-Eisner.

COMPLEMENT FIXATION TEST — In an affected person tuberculous antibodies are present in the serum Serum is mixed with tuberculin (as antigen) and tested for deviation of complement Results promising but still under trial

AGGLUTINATION Method of little value

No specific reaction at present can overweigh the results of clinical examination

X-Rays.—

In Normal Chest Outside the shadow of mediastinal contents is a shadow in position of hilus, with branches passing into the lung mainly due to blood in the pulmonary vessels

In Diseased Chests Shadows at and radiating from hilus increased ascribed to calcified glands and increased fibrous tissue Changes at site of apex slight No distinction between active and arrested disease Interpretations of shadows must be made with caution

Lagging of the Diaphragm (Williams' sign) — The diaphragm on affected side may move less than normal Of little use in early diagnosis Ascribed to pleurisy at base or possibly at apex involving phrenic nerve

TREATMENT OF CHRONIC PULMONARY TUBERCULOSIS.

Phthisis frequently heals but in the majority of such instances no symptoms have been recognized Evidence of recovery

Pathological Lesions found post mortem (i) Puckered scars at apex (ii) Calcified masses also (iii) Cavities with cicatrized walls (iv) Encapsulated caseous areas healing incomplete (v) Possibly pleural adhesions

Clinical —Recovery may occur after presence of tubercle bacilli in sputum

Measures of Progress.—Even with slight lesions constitutional disturbances are marked and these form the indications for treatment, and the measures of progress, rather than do the physical signs —

1 TEMPERATURE Of greatest importance (*see* SYMPTOMS) During treatment to be taken on waking about midday late afternoon and at bedtime
2 WEIGHT Recorded weekly
3 PULSE
4 PHYSICAL SIGNS
5 PRESENCE OR DISAPPEARANCE of tubercle bacilli in sputum

Indications for Treatment.—

Increase general resistance most important and only curative measure

Adopt measures against tubercle bacillus and tuberculous process.

Treat symptoms and complications.

Tuberculosis—Chronic Pulmonary—Treatment, *continued.*

General Treatment.

To Increase the General Resistance.—Essential methods of treatment are: (1) Open air; (2) Diet; (3) Rest and regulated exercise. These form the methods for the treatment of ordinary cases of phthisis. All others are subsidiary.

OPEN AIR.—Of first importance. Windows in room open day and night, sufficient for plentiful fresh air: not shut for cold or rain, or fever, cough, or other symptoms. Patient must never be cold, and must be warmly but not heavily clothed.

Unnecessary and even inadvisable to sleep outdoors or in shelters; but daytime to be spent there when allowed up.

DIET.—Diet larger than normal, especially in fat, but forced feeding (Debove) prejudicial.

MEALS.—Three, chief in middle of day. Nothing between meals. Rest of one hour before each meal essential.

DIET.—Mixed and varied: meat at each meal: plenty of potatoes, suet, and pastry. Fruit and green vegetables less important.

MILK.—One pint with each meal.

ALCOHOL.—Forbidden as routine. As stimulant for feeble pulse, and high temperature, or in dyspepsia.

RAW MEAT.—Is easily digested. Raw meat juice especially valuable in reduction of diet for alimentary disturbances.

GAIN IN WEIGHT.—Weekly gain desirable, 12 to 20 ounces: may be more for first weeks of treatment, afterwards rapid gain inadvisable: regulated by reducing milk. Too fat patients do badly. Final weight after treatment aims at one stone above previous best weight.

REST: REGULATED EXERCISE. Treatment divided into two periods: (1) Rest: (2) Regulated exercise.

PERIOD OF REST. If rectal temperature on coming under observation reaches 100° to 100·5° after one hour's rest, then rest in bed must be absolute (typhoidal rest). Allowed up when rectal temperature not above 98·6° for three successive days. Period of rest often many months.

PERIOD OF REGULATED EXERCISE. Amount of exercise strictly prescribed. Commences with few yards on level, slowly, without talking: distance gradually increased: then on slight incline. Further period of light work, as in a garden, systematically increased. After many months progress, walks may become 10 to 12 miles daily.

At all stages—Rest of one hour before meals.

Temperature.—Is guide to progress of exercise. After rest of one hour following exercise, must not exceed 98·6° in rectum: if so, reduce exercise or return to bed. If temperature continues to rise after exercise, severe auto-inoculation has occurred.

Other General Methods of Treatment.

DRUGS.—As little as possible: only with definite indications.

Most useful: –

COD-LIVER OIL.—Indications: (1) Full diet unobtainable (e.g., working men); (2) Weight not being gained on full diet. Give *small* dose, ʒj to iv, after meals; stop if causing nausea; combines well with malt.

MALT EXTRACT.—Good effect in weak digestion.

ARSENIC.—Specially if anæmia marked.

SANATORIUM.—Recommended for all early cases, and routine rigorously enforced. Duration of stay according to progress: not less than six months to a year or more. If stay limited to three months, routine learned must be carried on afterwards.

CLIMATE.—Now considered of secondary importance, although change of climate often does good. Main types are:—

SEA COAST.—Inadvisable for ordinary phthisis. Suitable for chronic cases with catarrh or much emphysema. Places: South Coast of England, Bournemouth, Torquay, etc.; Florida; Canary Islands, Madeira, West Indies.

DRY WARM CLIMATES.—Very suitable for all types if sand and dust avoidable. Places: South California; Algiers (Biskra); Egypt (Assouan, Luxor).

MODERATE ALTITUDES.—Woodlands Numerous and easily accessible; best for ordinary phthisis. Places (among many equally good): New Forest, Scotch Highlands; Adirondacks, Pyrenees

HIGH ALTITUDES.—

Advantages: Sunshine, pure air, equable temperature.

Disadvantages: Increased respiratory movements tend to produce emphysema, disadvantage on subsequent return to low level. also such movements may spread the disease in the lungs.

Unsuitable: Much emphysema.

Places (among many others): Davos, St Moritz, Arossa, Colorado Springs, Arizona.

Measures directed against the Tubercle Bacillus and Tuberculous Process.

Tuberculin. Numerous preparations exist: following are most important:—

KOCH'S OLD TUBERCULIN. Contains exotoxins. Six weeks culture of tubercle bacilli in 5 per cent. glycerin broth: evaporated to one-tenth volume: sterilized by heat: filtered. Initial dose ·00001 c.c. to ·0001 c.c.

KOCH'S TUBERCULIN R. (*Ruckstand*, residue or precipitate).—Contains endotoxins. Extracted from macerated bacilli: 1 c.c. contains extract from 10 milligrams of tubercle bacilli. Initial dose ·00001 c.c.

KOCH'S TUBERCULIN B.E. (*Bazillen Emulsion*).—A suspension of pulverized bacilli in glycerin and water. 1 c.c. contains 5 milligrams of bacilli. Initial dose ·00001 c.c.

T.R. AND B.E. most used for treatment: choice immaterial.

Tuberculosis—Chronic Pulmonary—Treatment, *continued*

INDICATIONS —Local lesions without constitutional disturbances are most suitable Fever is proof of auto inoculation, amount being irregular, variable and unmeasurable, and tuberculin is contra indicated by *rectal temperature above* 100°,

DOSE Commence with dose quoted and increase very slowly, e g, ·00001, 00002, 000035, 00005, 00007, 0001 Hypodermic injection

INTERVAL BETWEEN DOSES —Ten to fourteen days

CONTROL OF DOSAGE By temperature. No rise is desirable General rules —

1 No rise after injection Dose may be increased
2 Rise about 0 5° Repeat dose If similar rise again follows diminish dose
3 Rise 0 5° to 1° Return to small dose
4 Rise above 1° Abandon tuberculin at least temporarily

DANGERS —Large rise of temperature following injection may become continuous and patient worse ascribed to reaction at focus Such rise may suddenly follow small increase or repetition of previous dose an anaphylactic phenomenon Great caution always necessary, each dose being considered carefully Use of tuberculin in pulmonary tuberculosis is now almost abandoned

Drugs —None has specific action Chief use of drugs is in treatment of symptoms

CONTINUOUS INHALATION Good results published but method not in general use Tends to impede respiration Turney Yeo's inhaler worn continuously except at meals Add every half hour a few drops of inhalation e g

R Creosoti		Spt Chloroformi	
Ir Iodi	ana ʒj	S V R	ana ʒiss
Ol Eucalypti			

Artificial Pneumothorax.

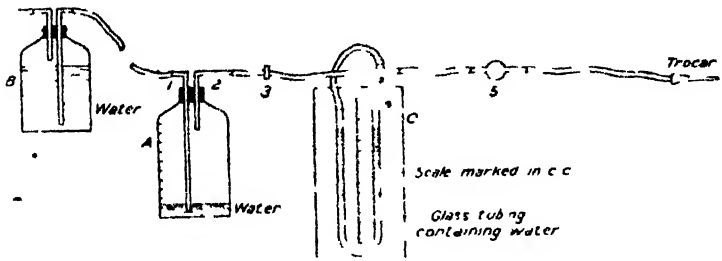
Suggested by good results occasionally following spontaneous pneumothorax, by resting diseased lung

INDICATIONS Chronic unilateral disease, especially when advancing in spite of treatment. Valuable in uncontrollable hæmoptysis. Bilateral disease contra indicates

METHOD Bottle *A* capacity 1000 c.c., graduated each 100 c.c. on outside, filled with nitrogen from a cylinder through rubber stopper pass two glass tubes connecting by rubber tubing (1) with bottle *B*, containing water and standing feet above *A* and by (2) with trocar. Between *A* and trocar are (3) a clip on rubber tubing (4) a T piece connecting with water manometer *C* registering pressures up to 30 c.c. water, and (5) a glass bulb containing cotton wool to filter nitrogen When clip is loosened water flows by siphonage into *A*, and this pressure is used to drive nitrogen into pleural cavity Whole apparatus is sterilized

TECHNIQUE —No anæsthetic necessary With clip closed, trocar

is inserted into chest, presence in pleural cavity being shown by oscillations of manometer. On first treatment (negative) pressure in pleura will be 8 to 12 c.c. water. On opening clip gently, water flows from *B* to *A* and nitrogen enters pleural cavity.



Continue slowly until pressure has increased 1 c.c., viz. 4 to 5 c.c. water; volume used from 100 to 500 c.c. on first occasion. Withdraw trocar and seal puncture.

In first year repeat about every time; until intrapleural pressure will be about 1 c.c. higher each time. Then lengthen intervals between injections. At each injection raise pressure 1 c.c. above commencing pressure. After three months, pressure about 20 c.c. water being sufficient to collapse lung against spine. Should not exceed 25 c.c. Subsequently interval between injections about six weeks, watching physical signs of pneumothorax.

RESULTS. Very successful in selected cases. Duration of treatment one to two years. Pleural adhesions may render proceeding impossible; otherwise usually simple, especially after first injection. Shock may occur if too much gas be injected at once.

Treatment of Symptoms and Complications.

Fever. Should rarely be interfered with directly; best treated by rest (see GENERAL TREATMENT). Above 103 tepid sponging may relieve. In later hectic stages reduction of temperature usually fails to improve patients. All drugs to be used with caution; best phenazone, acetanilide, quinine.

Night Sweats. Sponge at bedtime with vinegar and water, or with alcohol (Eau de Cologne), rub dry. If distressing, pill of ext. belladonnæ gr. ¼, and zinc. oxid. gr. ½ to iij. Flannel night clothes, to absorb moisture.

Cough. Routine use of expectorants prejudicial. LOOSE COUGH, bringing up sputum, is beneficial. Trochiscus glycyrrhizæ (Brompton cough lozenge) is valuable —

℞ Ext. Glycyrrhizæ gr. iij; Tinct. Aconiti gr. x
Olei Anisi ♏ ss.
Occasionally.

Tuberculosis—Chronic Pulmonary—Treatment, *continued.*

DRY HACKING COUGH.—Often under patient's control. Patient taught to hold breath and urged to restrain cough.

IRRITATING USELESS COUGH.—Determine the cause, e.g. laryngitis. Sedatives often necessary, especially for nocturnal cough.

a. Try warm saline drinks, e.g. sod. bicarb. gr. xv in glass of water. Often effectual for morning cough, if taken on waking.

b. Sedatives: morphia, codeine, or heroin:—

℞ Liq. Morph. Hyd.	♏xx	Syr. Limonis	♏xx
Acid. Hydrocyan. Dil.	♏iij	Glycerini	ad ℥j

Or,

℞ Syr. Codeinæ Phosph.	♏xx	Spt. Chloroformi	♏ij
Syr. Aurantii	♏xx	Glycerini	ad ℥j

Or,

Heroin gr. ¼ (for linctus *see* BRONCHITIS)

Or, if severe,

Omnopon gr. ¼

SPUTUM TENACIOUS.—Expectorants temporarily: unguentum ammon. chloride. Lin. terebinth. aceticum, rub on chest night and morning.

SPUTUM EXCESSIVE. Terpene hydrate gr. ij to v, t.d.s. or creosote in capsules ♏ij, t.d.s. or eucalyptus oil in capsules ♏v, t.d.s.

RECURRENT BRONCHITIS. Sometimes improved by autogenous vaccines of predominant bacteria.

Linimentum Terebinthi Aceticum may ease tight cough.

Gastro-intestinal Disturbances.

ANOREXIA.—Bitters before meals, or mixture.

℞ Sod. Bicarb.	gr. xv	Tm. Chloroformis	♏x
Tr. Nux Vom.	♏v	Inf. Gent. Co.	ad ℥j

Acids sometimes effective: also pepsin and ferments.

DYSPEPSIA.—As above, or bismuth often valuable.

℞ Bismuth Oxycarb.	gr. xx	Mucil. Tragacanth	♏xv
Sod. Bicarb.	gr. xx	Aq. Chloroformi	ad ℥j
Acid. Hydrocyan. Dil.	♏iij		

t.d.s.

Reduced diet may be necessary; raw meat juice is then valuable.

DIARRHŒA.—Should never be neglected: often troublesome, and rapidly debilitating.

LIGHT DIET. WARMTH to abdomen and extremities. BED if necessary.

DRUG:—

1. Dover's powder, gr. x t.d.s., usually best.

2. Coto and opium--

℞ Tr. Coto ♏xv | Tr. Opii Sed. (Battley) ♏xj
Aq. ad ℥ss

3. Acid diarrhœa mixture.
4. Bismuth.
5. Enema opii.

Pain.—If muscular from strain of coughing, rub with liniment of aconite, belladonna, and chloroform.

For pleurisy: strap side, unless other lung is tuberculous, when counter-irritation should be employed.

Insomnia.—Warm drink at night. Sedatives for cough. Drugs: Trional and chloralamide gr. xx ãã.

Dyspnœa.—Rarely severe, except in late stages or very rapid progress. Stimulants, e.g., ammonia and ether.

Hæmptysis.—*See* DISEASES OF THE RESPIRATORY SYSTEM.

Pneumothorax.—*See* DISEASES OF THE RESPIRATORY SYSTEM.

IV. TUBERCULOSIS OF THE LYMPHATIC GLANDS.

(*Scrofula. Tuberculous Adenitis.*)

Etiology.—

AGE.—Most common in children, but no age immune.

PREDISPOSING FACTORS.—Catarrh of mucous membranes: (a) Enlarged tonsil, adenoids, carious teeth (adults), for cervical glands; (b) Whooping-cough, measles, for bronchial glands.

Two physical types formerly emphasized: (1) Tall, slight, intelligent, with clear skin; (2) Thick set, coarse and dull.

Site.—*Cervical glands* most common, often no other lesion: especially from three to ten years. In infancy, often deeper glands and more widespread.

Morbid Anatomy. Ordinary changes of tuberculosis modified histology of glands (*see* HISTOLOGY OF TUBERCULOSIS, p. 125).

Tubercles commence in cortex: gland enlarges: on section gray tubercles visible. If very early may be none visible, but microscopically foci of epithelioid cells and leucocytes. *Tubercles increase*, distinction between medulla and cortex lost, and usual changes follow, viz.: (1) Fibrosis. (2) Caseation. (3) Calcification. Often proceeding simultaneously. Gland on section in such stages is pale and homogeneous with caseous areas, gritty in places, and with capsule thickened. Also (4) Softening: common in superficial glands, producing 'cold abscess'. Periadenitis common, *adherence to other glands and structures.* Thus termination may be: (i) Calcification, especially deep glands; (ii) Dry caseous mass with fibrous capsule; (iii) Softening and rupture.

SPLEEN may be enlarged.

Tuberculosis of the Lymphatic Glands, *continued.*

Noticeable Features.—In tuberculous adenitis: (i) Local character, often confined to one series of glands, e.g., cervical, (ii) Spontaneous healing common, e.g., calcified mesenteric glands; (iii) Frequent suppuration, especially in cervical glands, (iv) Glands may be long quiescent, and then cause acute miliary tuberculosis owing to adhesion to, and infection of, blood-vessels, and subsequent spread of tuberculosis through the blood.

Local Tuberculous Adenitis.—Of palpable glands, cervical group is most common seat, then axillary; glands of groin rarely.

CERVICAL.—Especially glands in posterior cervical triangle: commencing in glands at level of jaw, spreading down neck (superficially and deeply), then glands above clavicle, and axillary glands. Submaxillary glands common. Usually larger on one side than the other.

SYMPTOMS.—Anæmia, slight pyrexia if progress rapid.

PHYSICAL SIGNS.—In early stages, glands discrete; later form an adherent mass from periadenitis. Subsequently adherent to skin, and finally soften and rupture if untreated.

Note Palpate cervical glands from behind

PROGRESS.—Slow, often persist many years. Death rare

TRACHEOBRONCHIAL Common site, bifurcation of trachea, on right side.

SYMPTOMS.—*Cough*, paroxysmal and brassy (not unlike whooping-cough). Also wasting, anorexia, slight fever

PHYSICAL SIGNS Absent or indefinite unless large mass May be: (i) Dullness over manubrium sterni, (ii) Distended veins in neck; (iii) Venous hum over clavicle, *audible only on retracting head* (ascribed to pressure on veins), (iv) Signs of pressure on bronchi. It is said that the resulting pulmonary collapse may simulate phthisis, especially apical, viz., impaired resonance and feeble breathing but no râles.

X-RAY.—Shadow may be distinct.

PROGRESS AND SEQUELÆ.—(i) *Healed calcified glands* or quiescent encapsulated caseous glands are common post mortem. (ii) *Acute miliary tuberculosis*: through adhesion and discharge into vessels. (iii) *Infection of the lung or pleura* i.e., pulmonary tuberculosis. Hilus or base of the lung may be affected earliest: usually in children. Rare are (iv) Pressure on vessels or nerves (recurrent laryngeal) (v) Perforation into bronchi, trachea, œsophagus.

TABES MESENTERICA.—Mesenteric and retroperitoneal glands affected. Mostly in childhood. Especially common in Scotland. Often primary, but may be associated with tuberculous peritonitis, either as cause or effect.

SYMPTOMS.—Wasting and debility. Puny limbs. Diarrhœa rarely absent. Fever moderate. Abdomen distended and hyper-resonant. Glands may form large mass, usually to right of umbilicus, but palpation often difficult owing to distention. Glands very rarely suppurate and rupture. Calcified quiescent caseous glands frequent at autopsies.

Generalized Tuberculous Adenitis.—Not common. Widespread tuberculosis of lymphatic glands; other tissues little or no affection. Deep glands most involved, retroperitoneal, mesenteric, and bronchial, but superficial groups may also be affected. May be great enlargement. Spleen may be palpable. Temperature high but irregular. Constitutional symptoms severe but often indefinite. Polynuclear leucocytosis may be marked. Death from cachexia or, in children, not uncommonly tuberculous meningitis.

Diagnosis of Tuberculous Glands. In children usually simple, in adults often difficult. Diagnosis from—

HODGKIN'S DISEASE. Chief diagnostic difficulty; important owing to high mortality. Note: (1) Glands remain discrete in late stages may be some adhesions from periadenitis, but never adherent to skin and never soften. (2) Spleen usually palpable. Definite diagnosis impossible without microscopic section of gland.

PYOGENIC ADENITIS. Secondary to septic focus on scalp, etc.

GLANDULAR FEVER.—Rapid enlargement and subsidence.

SYPHILIS.—Enlargement general and usually slight; subsides with antisyphilitic treatment. Wassermann reaction positive. Other signs of syphilis.

SECONDARY MALIGNANT DEPOSITS. Primary growth elsewhere. Microscopic section.

BLOOD DISEASES. Examination of blood.

LYMPHOSARCOMA. Rapid growth and early adhesions.

Rare are

CYSTIC HYGROMA.—Congenital } Fluctuation. No surrounding
BRANCHIAL CYSTS. Position } glands.

Blood Changes. Leucocytosis with relative lymphocytosis, similar to Hodgkin's disease. In generalized form, polynuclear leucocytosis, both in absolute numbers and relative percentage.

Spleen. Occasionally but not usually palpable. (In Hodgkin's disease enlarged in over 70 per cent.)

Treatment.

EARLY STAGE, GLANDS DISCRETE—

1. REMOVE LOCAL INFECTION. Teeth, tonsils, and adenoids.
2. GENERAL TREATMENT. Sea air especially Margate or a voyage. Tonics, cod-liver oil.
3. LOCAL.—Paint (not rub) glands with iodine ointment. Less important.

GLANDS SOFTENING OR INCREASING after three to six months' treatment.—Operate, and follow with above treatment.

X RAYS.—*Have undoubted and valuable therapeutic effect.*

Tuberculosis of the Lymphatic Glands—Treatment, *continued*

TUBERCULIN.—Good results follow, with small doses at fortnightly intervals over long periods Other treatment must not be sacrificed. (*See* CHRONIC PULMONARY TUBERCULOSIS, p. 153)

V. TUBERCULOSIS OF THE PERITONEUM.

(*Tuberculous peritonitis*)

Etiology.—

AGE —Specially in young children: frequent up to 20 years then becomes progressively less frequent, but occurs at all ages.

SEX.—In adults, commoner in women than men owing to infection from Fallopian tubes

MODE OF ORIGIN —

1. Most frequently no cause found tubercle bacilli of bovine type in 25 to 50 per cent, hence milk accepted as origin, bacilli passing through mucous membrane without causing lesion
2. From Fallopian tubes common cause in women
3. Pulmonary tuberculosis: sputum swallowed, may or may not be intestinal ulcers

Occasional origins and associations —

4. Pleura (and rarely pericardium) may be also affected constituting polyorrhomenitis
5. Primary tuberculosis of intestine
6. Occasionally from the vesiculæ seminales Frequently terminal in cirrhosis of liver Not uncommon with ovarian tumours. May occur in hernial sacs

Morbid Anatomy.—Gray tubercles may be present on the peritoneum in acute miliary tuberculosis, pulmonary and generalized, also in chronic pulmonary tuberculosis, and on peritoneal surface of tuberculous ulcers of the intestine In the more widespread disease clinically constituting tuberculous peritonitis', the following tissues may be concerned —

PERITONEUM —May be: (i) Tuberculous masses in peritoneum, often caseating: *the omentum is frequently involved.* (ii) Peritoneal adhesions between coils due to fibrosis. (iii) Diffuse peritonitis tubercles scattered over peritoneum. specially associated with ascites

MESENTERIC GLANDS.—As in tabes mesenterica (*See* p 158)

INTESTINAL MUCOUS MEMBRANE.—Often but not always tuberculous. affects symptoms but not physical signs

Results of Lesions.—The changes just mentioned may exist and co-exist to varying extents; and depending upon this are the following common results —

1. PRESENCE OF AN ABDOMINAL TUMOUR —May be —

1. OMENTUM —Tuberculous and 'rolled up'. palpable, lying across abdomen near umbilicus common.

ii SACCULATED EXUDATION —Due to combination of effusion and adhesions between coils position usually central, and may simulate ovarian tumour

iii ENLARGED GLANDS May form large masses

iv INTESTINAL COILS Thickened Rarely palpable

v FÆCAL ACCUMULATIONS Extremely common, from intestinal obstruction and interference with peristalsis Removed by enema

Failure to feel masses subsequently found at operation may be due specially to (a) Ascites, (b) Tympanites

2 ASCITES Specially with diffuse peritonitis also affected by enlarged glands in hilus of liver

3 ADHESIONS BETWEEN COILS from fibrosis May be extreme tuberculous ulceration of intestine may lead to perforation into another different coil, or to formation of localized abscess

4 FISTULÆ May form from extension and caseation of tuberculous masses usually at umbilicus may be fæcal if also ulceration and adhesions of intestine

5 TYMPANITES Due to (i) Peritoneal adhesions in chronic cases, (ii) Loss of tone in acute cases

Clinical Groups. Two types (i) Ascitic (ii) Plastic

ASCITIC TYPE Characterized by large amount of fluid

PLASTIC TYPE Amount of fluid small Tumours and irregular masses common

1 ULCERATIVE Tuberculous masses in peritoneum caseation may result in abscesses and fæcal fistulæ some fibrosis and hardening of intestines

2 FIBROUS Adhesions between coils marked little fluid Very chronic intestinal obstruction may result For extreme form see CHRONIC PROLIFERATIVE PERITONITIS and TUBERCULOUS CÆCAL TUMOUR

3 TABES MESENTERICA Masses of tuberculous glands Other changes slight

Symptoms.

MODES OF ONSET (a) Latent (b) Disease is most frequent, slight pyrexia and abdominal tenderness and distention may resemble typhoid fever temporarily (c) Acute suggesting appendicitis or intestinal obstruction

SYMPTOMS —

SLIGHT GENERAL DISTURBANCE Weakness, loss of weight, pallor

FEVER Variable In commoner chronic cases slight about 100, either continuous or at intervals frequently subnormal In acute form often 103 to 104

GASTRO-INTESTINAL SYMPTOMS Usually no vomiting or nausea Constipation, if no ulceration of intestine Diarrhœa, if intestine ulcerated generally only looseness may be offensive stools

PAIN Usually slight • may be paroxysms due to obstruction Tenderness on pressure

Tuberculosis of the Peritoneum—Symptoms, *continued.*

PIGMENTATION.—Abdominal, or even general, not uncommon. Buccal mucous membrane unaffected (*cf.* ADDISON'S DISEASE).

Physical Signs.—Vary with type: intermediate forms frequent.

ASCITIC TYPE.—*Abdomen greatly distended*, shifting dullness in the flanks. Adhesions may cause sacculated exudations in more chronic cases.

PLASTIC TYPE. (i) Ulcerative form: common variety: abdomen moderately distended, with characteristic doughy feel, no visible peristalsis: may be no other signs, but commonly *indefinite masses*, from omentum, glands, or tuberculous matter between coils. (ii) Fibrous form, palpable tumour, may be irregular, coils, rolled-up omentum, or fæcal accumulations. (iii) Palpable tuberculous glands (*tabes mesenterica*): tumour central or near cæcum, usually fixed, outline irregular, hard.

Course.—In unfavourable cases, progressive wasting with increasing abdominal signs.

Diagnosis.—

IN CHILDREN.—Usually simple: suggested by the following: (1) Ascites—rarely due to other causes. (2) Progressive loss of weight with slight disturbance of bowels; or (3) Prolonged slight diarrhœa and slight pyrexia.

IN ADULTS. Often difficult. Diagnosis from:—

OVARIAN TUMOUR. No fever, no shifting dullness, outline definite, tumour usually central, no disease in lungs, pleura, or Fallopian tube.

CIRRHOSIS OF LIVER. History and appearance of patient, cytology of fluid on paracentesis if shifting dullness; edge of liver may be palpable.

MILIARY CANCER WITH ASCITES.—Cytology of fluid.

PERITONEAL FLUID.—For characters *see* PLEURAL FLUIDS.

Treatment.—

MEDICAL.—

GENERAL.—Complete rest until afebrile: sea air, especially Margate.

DIET.—Full diet with fat. In diarrhœal cases, milk diet.

LOCAL TREATMENT. Inunctions of mercurial ointment once daily. Commence for child with gr. viij, rising to gr. xxx: continue for weeks but intermit if diarrhœa.

TONICS.—Cod-liver oil. If anæmia, syrup of the iodide of iron.

DIARRHŒA.—Check with Dover's powder, or with bismuth or chalk with tr. opii (♏j for each year in a child—Hutchison).

SURGICAL.—Ascitic cases, with pyrexia and not subsiding under above treatment, frequently improve after laparotomy. Plastic type unsuitable for operation.

Prognosis.—*Good*, generally, in plastic type and also in ascitic type if fluid diminishes with rest. *Bad*:—(1) Under 2 years: tuberculosis nearly always becoming generalized. (2) With tuberculous enteritis. (3) After formation of fæcal fistulæ.

VI. TUBERCULOSIS OF THE ALIMENTARY CANAL.

LIPS Very rare Occasionally ulcers from infection by sputum

TONGUE Occasionally ulcers present in late stages of phthisis Tuberculous sputum may infect crack and ulcer form

CHARACTER OF ULCER On dorsum or edge usually small irregular edge and gray floor may be multiple extremely painful

DIAGNOSIS Tuberculosis of lungs or larynx present Diagnosis from (1) Syphilis Wassermann reaction (2) Cancer excise portion and examine

TREATMENT Lunt with cocaine before meals

SALIVARY GLANDS Practically immune

PALATE Only by direct extension

TONSILS Groups (1) Tuberculous ulceration Rare Infection from sputum Never primary (2) Enlarged tonsils microscopically tubercles found (3) No macroscopic or microscopic tubercle, but inoculation produces tuberculosis in animals Frequency still doubtful important point for observation in etiology of tuberculosis of glands and lungs

PHARYNX By extension from the larynx or TUBERCULOUS LARYNX

OESOPHAGUS Of doubtful occurrence

STOMACH Of doubtful occurrence Immunity ascribed to acidity of contents

FISTULA IN ANO Common in tuberculosis

VII. TUBERCULOSIS OF THE INTESTINES.

Groups. (1) *Primary* (2) *Secondary to phthisis* (3) Secondary to tuberculous peritonitis A special type of *ulceration* *tuberculosis of the ileocaecal region*

Morbid Anatomy.

SITE Ileum, caecum, and colon Most commonly at end of ileum Commences in Peyer's patches and solitary follicles By small tissue undergoes tuberculous change swelling caseation softening and ulceration

CHARACTER OF TUBERCULOUS ULCER (i) *Shape.* spreads *transversely round gut* by lymph ducts and blood vessels may encircle it (ii) *Walls and edges* thickened and raised but not undermined contrasting with typhoid ulcers (iii) *Floor* caseous, often shows miliary tubercles (iv) Peritoneal surface thickened and miliary tubercles present All layers are involved Ulcers may be multiple

Primary Intestinal Tuberculosis. Extremely rare except in children Infection by milk

Symptoms (i) Irregular bowels diarrhoea or constipation, (ii) Pyrexia (iii) Pains (iv) Wasting Recurrent attacks may occur, closely simulating appendicitis

Rarely diagnosed until sequelae appear or tuberculosis in lungs or meninges

Tuberculosis of the Intestines, *continued*

Secondary Intestinal Tuberculosis. Due to swallowing sputum. Present in 50 to 70 per cent of autopsies in phthisis

Sequelæ.—(i) *Perforation and peritonitis* uncommon owing to thickening of peritoneum and formation of adhesions (ii) *Localized abscess formation* following perforation (iii) *Stenosis of intestine* may result from fibrosis and cicatrization of healing ulcer in small intestine with fluid contents obstruction may be slight (iv) Tuberculosis of mesenteric glands and peritoneum common in primary type (v) *Hemorrhage* very rarely serious but is occasionally fatal

Diagnosis.—

PRIMARY TYPE In children Fever, irregular bowels wasting may be palpable glands

SECONDARY TYPE Diarrhœa in course of phthisis but this may also be due to catarrh or tuberculous disease

Tubercle bacilli are present in stools in both forms

Treatment. See DIARRHŒA IN PULMONARY TUBERCULOSIS (p. 150)

Hyperplastic Tuberculosis of the Ileocæcal Region (*Tuberculous Cæcal Tumour*) Uncommon but important condition Great thickening of the muscular and fibrous coats walls may be one inch thick lumen greatly diminished In rare cases may affect sigmoid or small intestine

PATHOGENESIS The condition merges into CHRONIC PROLIFERATIVE PERITONITIS (q.v.) and the tuberculous origin of the group is uncertain

TYPES (1) Definite tumour in right iliac fossa tuberculous cæcal tumour usually vertical hard fixed and tender (2) General thickening in right iliac fossa but no definite tumour as in recurrent appendicitis Faecal fistula may form

SYMPTOMS. Attacks of pain in right iliac fossa irregular diarrhœa and constipation pyrexia and wasting

DIAGNOSIS From:

CANCER Difficult even at operation Duration longer than in cancer, usually two to three years, but may be longer age generally under forty years

APPENDICITIS Second type especially simulates appendicitis diagnosis before operation may be impossible accounts for certain cases of obstinate fæcal fistula seen after appendix operations Tubercle bacilli may be present in discharge or even in stools

DIVERTICULITIS

TREATMENT Operation, lateral anastomosis and removal of tumour if possible complete removal of all tissue is not essential

VIII. TUBERCULOSIS OF THE LIVER.

Rare. Occurs in various forms:—

1. MILIARY TUBERCULOSIS.—Common in acute miliary tuberculosis. Tubercles in capsule and tissue: usually scanty and very minute, especially in tissue.
2. SOLITARY TUBERCLE. Tuberculous mass. Very rare.
3. TUBERCULOUS PERIHEPATITIS AND CIRRHOSIS. Occurs with chronic proliferative peritonitis. No absolute proof of tuberculous origin.
4. TUBERCULOSIS OF GALL-BLADDER AND BILE DUCTS. Very rare. Occasionally recorded with gall stones.

IX. TUBERCULOSIS OF THE PLEURA.

Very common. May be (*a*) Primary, (*b*) Secondary.

Primary.

ACUTE PLEURISY. Unknown how frequently a primary dry pleurisy is tuberculous. (Probably a primary pleurisy is nearly always tuberculous.)

PLEURISY WITH EFFUSION. A primary clear pleural effusion is practically always tuberculous. Often terminates in pulmonary tuberculosis. (*See* PLEURISY WITH EFFUSION.)

CHRONIC ADHESIVE PLEURISY. Great proliferation and thickening of the pleura, usually with cirrhosis of the lung. (*See* CHRONIC INTERSTITIAL PNEUMONIA.)

POLYSEROSITIS (*Polyarthromeningitis*). General proliferation of serous membranes, peritoneum, pericardium, etc. Doubtful if all forms are tuberculous. (*See* CHRONIC PERITONITIS.)

Secondary. Chronic pleurisy and adhesions almost invariable to some extent in chronic pulmonary tuberculosis. Pleurisy with effusion very common; may be clear, hæmorrhagic, or purulent. Onset may be acute or insidious.

Pyopneumothorax not infrequent.

X. TUBERCULOSIS OF THE BRAIN.

Occurs as (1) *Acute miliary tuberculosis*, viz., tuberculous meningitis. (2) *Solitary tubercle*, produces symptoms of cerebral tumour; the meninges are usually affected to some degree. (*See* INTRACRANIAL TUMOURS.)

Section I – Specific Infectious Diseases, *continued*

B—NON-BACTERIAL FUNGUS INFECTIONS.

CHAPTER XIX.

ACTINOMYCOSIS. MADURA FOOT.

ACTINOMYCOSIS.

A chronic suppurative disease produced by the *Streptothrix actinomyces* or ray fungus.

Occurrence in Nature. Occurs mainly in cattle, horses, and man; occasionally in pigs. Summary of main differences between cattle and man. (1) *Cattle*: Especially affects jaw and tongue, causing large, hard swellings (woody tumor); bone, jaw very chronic; formation of much granulation and fibrous tissue; Clubs numerous and Gram positive; filaments less definite. (2) *Man*: More rapid, greater tendency to suppuration and less to granulation tissue formation; swellings smaller and less hard; microscopically, clubs fewer or absent and Gram negative; filaments numerous.

The following [illegible] occurring in MAN will be specially noted.

The Parasite. A streptothrix characterized by true branching (hence not bacterial); probably allied to *Nocardia*.

MORPHOLOGY: Heteromorphous. Three elements present in tissues:

- FILAMENTS: Thin, length indefinite, *true branching*; Gram positive. In old colonies often dense and irregular strands.
- SPORES OR GONIDIA: Gram positive; coccoid forms; can reproduce filaments.
- CLUBS: Hyaline swelling of extremity of filament; probably structureless pear-shaped body; may form a ring at edge of colony; probably defensive against phagocytes. In man often absent, often Gram negative. (In cattle numerous and Gram positive.)

CHARACTER OF PUS: From an abscess pus is thin greenish yellow, *containing small clumps size of a pin's head* visible to the naked eye, usually yellow and formed of the streptothrix. (Old pus may be thicker.)

Examination of Pus: Pick out clump and place on microscope glass; tease or squeeze clumps flat between cover slips; stain with Gram or carbol thionin. Branching filaments easily recognized.

CULTIVATION.—Difficult. *Anaerobe* • (J. H. Wright and others.)

MODE OF INFECTION.—Formerly supposed to be present on grain, especially barley. Proof of anaerobic nature now renders this doubtful. Probably direct infection from animals. Still under discussion.

Changes produced in Tissues. Chronic inflammatory reaction, little granulation tissue, spreading suppuration, mainly filamentous forms. In dense tissues more granulation, in soft tissue, e.g., liver, more pus formation.

Clinical Characteristics. Usually in males, commonly connected with cattle. Three chief sites, possibly corresponding to paths of infection.

JAW AND NECK.—*Chronic* unilateral swelling of jaw and neck, later superficial *abscesses* on skin, which discharge pus. Mode of infection probably through carious tooth or tonsil. Forms 50 per cent. of cases.

INTESTINAL.—Especially *appendix*, also caecum and large intestine. Great tendency to spread in various directions, to peritoneum, forming abscesses between coils, to abdominal wall, causing sinus and abscess, to retroperitoneal, retroceal and perirectal tissues. Occasionally in oesophagus. Infection probably with food.

SYMPTOMS.—Depend on site, septic phenomena, chronic appendicitis, often indefinite nature. Includes about 20 per cent. of cases.

3. PULMONARY.—Various types of chronic pulmonary disease: (a) Chronic bronchitis, (b) Resembling miliary tuberculosis, (c) Bronchiectasis, fibrosis, fatal bronchitis. Great tendency to involve pleura, ribs, sternum, and thoracic wall, forming abscesses. Infection respiratory, or possibly from mouth, oesophagus, or abdomen.

SYMPTOMS.—Irregular pyrexia, cough, wasting, sputum which may contain streptothrix. Physical signs often [illegible] lateral.

METASTASIS.—Formation of secondary abscesses occur in many directions, possibly by leucocytes engulfing filaments. Of importance are:

LIVER.—Not uncommon, characteristic honeycombed appearance, containing pus.

CEREBRAL ABSCESS (in head cases).

Skin infections have been recorded. Also kidneys, etc.

Diagnosis. Depends on identification in pus, tissues, or sputum of the causal streptothrix, viz., branching filaments or clubs. Suggestive: (1) Association with cattle and horses; (2) Chronic pyaemia; (3) Character of pus. Fairly clear with *jaw-head* masses.

Prognosis.—Depends on extent of spread and on site: (1) Jaw and neck: recovery usual. (2) Intestinal: prognosis fair. (3) Pulmonary: fatal. Always prolonged. Tendency to recurrence after apparent cure: but rare after two years' freedom.

Actinomycosis, *continued.*

Treatment. Combined surgical and medical.—

SURGICAL. Treat on general principles usually impossible to remove focus entirely.

MEDICAL. Potassium iodide in maximum doses (gr xl to lx daily) for many months.

X rays in superficial forms

Variations.

Actinobacillus A small Gram-negative aerobic bacillus has been isolated from cattle in conditions resembling actinomycosis but in which no filaments were present Is pathogenic to animals, clubs being subsequently present in the lesions

A *streptothrix*, aerobic and liquefying gelatin, has been isolated repeatedly, but it does not reproduce lesions, and its presence is probably accidental.

MADURA DISEASE.

(*Madura Foot*)

A chronic granuloma practically confined to the foot and characterized by great enlargement, formation of cavities and discharge of granules Very prevalent in India and other parts of the tropics *Foot* almost invariable site of affection Rarely hand

Clinical Description.— Extremely chronic progress very slow

ONSET Swelling on foot irregular nodular character slowly softens in centre discharges *granules*

PROGRESS Great enlargement of foot numerous sinuses and cavities caries of bone Internal lesions never occur

CHARACTER OF GRANULES Granules are present in cavities larger than in actinomycosis collections form definite nodules Two varieties occur

1. *Pale* Is a streptothrix morphologically resembles actinomycosis, but is distinct, aerobic does not liquefy gelatin no effect on animals
2. *Black.*— Is a hyphomycete (J H Wright)

Treatment.—Excision or amputation Potassium iodide valueless X rays good results recorded Antimony, intravenously, under trial

Section I.—**Specific Infectious Diseases,** *continued*

C. PROTOZOAN INFECTIONS.

CHAPTER XX.

MALARIAL FEVER.

Conditions due to infection with certain specific protozoa conveyed by bite of mosquitoes, and characterized by fever, often periodic and controlled by quinine, occasionally by malignant fatal forms, and by a chronic anæmia and enlargement of the spleen.

Geographical Distribution. Almost universal where warmth and water exist together. Main areas:

EUROPE: Balkan States, Greece, South Russia, portions of Italy and Spain.

AMERICA: Southern States, Atlantic Coast of Central America, South America.

ASIA: India, Burma.

AFRICA: Practically all river basins.

THE PARASITE.

Three definite species exist in man: (1) Benign tertian, *Plasmodium vivax*; (2) Quartan, *P. malariæ*; (3) Malignant tertian—*P. falciparum*.

The *life cycle* has two stages: (1) *Intracorpuscular*. Asexual. Recurrent cycles in human body. Man is the intermediate host. (2) *Extracorpuscular*. Sexual. Single cycle in the mosquito, which acts as the definitive host.

1. Cycle in Human Body.* Intracorpuscular and asexual. Principal characteristics are: (a) Recurrent asexual cycles taking place in the red cells. (b) Production of sexual cells, of which the further development only takes place in the mosquito.

Cycle is commenced by entry of protozoon into red cells. Chief stages in cycle and growth of protozoon are:

1. THE PROTOZOON may enter the red cells either as:—
   - SPOROZOITE: Introduced by mosquito bite, adheres to and penetrates a red cell (primary infection). Or
   - MEROZOITE: Spore produced in human cycle penetrates red cell (recurrent cycle).

* Muir and Ritchie, *Manual of Bacteriology*, xi.

Malarial Fever—The Parasite, *continued*

2 TROPHOZOITE Growth of amœbula within red cells Protozoon grows in size exhibits amœboid movement pigment appears as granules and increases in amount forming from hæmoglobin

MORPHOLOGY (Leishman and other Romanowsky stains)

i Shape very variable in earlier stages often 'ring forms'

ii. Protoplasm stains blue

iii Intense red chromatin in nucleus

iv Dark pigment

Enlarged parasite no amœboid movement roughly round

3. SCHIZONTS *Stage of sporulation* Protoplasm divides into segments into which the red chromatin scatters pigment collects in centre Red cell ruptures dispersing the spores so called merozoites pigment enters leucocyte

4 MEROZOITES (spores) Round about 2 μ protoplasm stains blue red chromatin in nucleus Spore attaches to and enters red cell, and a fresh cycle commences

GAMETOCYTES Sexual cells

Certain trophozoites become sexual cells, dividing no further in man In *benign tertian and quartan* parasites resemble large full grown parasites' Two forms (a) Macrogametocytes, female cells, stain deeply, pigment granules (b) Microgametocytes male cells, smaller, stain more faintly less pigment central nucleus with chromatin In *malignant tertian* parasite 'Crescent' form

Varieties of Parasite. Main differences in human types

1 BENIGN TERTIAN *Plasmodium vivax*

Cycle forty eight hours

Red cells enlarged, pale, may be basophilic degeneration

Pigment fine light brown granules

In fresh blood active amœboid movements outline indistinct

Schizont rosettes 15 to 20 regularly arranged spores

2 QUARTAN *Plasmodium malariæ*

Cycle seventy two hours

Red cells unaltered in size and appearance

Pigment coarse dark brown granules

In fresh blood amœboid movement slight outline distinct

Schizont daisy heads 6 to 12 regularly arranged spores

Somewhat smaller than benign tertian

3 MALIGNANT TERTIAN (Estivo autumnal) *Plasmodium falciparum*

Main distinctions from previous forms are

(1) Sporulation and great portion of cycle takes place almost entirely in internal organs, especially spleen In peripheral blood parasites scanty and are mainly sexual 'crescents' and asexual ring forms

(2) Gametocytes are 'crescentic' not resembling the asexual forms Only appear after several days fever 'Crescent' distinct outline, pigment and chromatin in centre

remains of red cell often visible Male form is fatter, stains lighter, and has pigment more scattered than female.

Other differences

Cycle uncertain Probably forty-eight hours
Red cells shrivelled and dark
Pigment very scanty, fine granules
Parasite small Amœboid movement active
Schizont (in spleen) 6 to 20 small irregularly arranged spores

2. Cycle in Mosquito. Extracorpuscular and sexual

Cycle commences from *gametocytes* taken into stomach of feeding mosquito asexual form from human blood take no part (The earlier stages will occur in a drop on a microscope slide)

1 DEVELOPMENT OF GAMETOCYTES

Male cell vibratile movements of pigment granules become visible then flagella are extruded forming flagellated body The flagella are long thin often with a bulbous end, and have a deep red chromatin core covered with protoplasm (Ross considers microgametes and not true flagella)

Female cell maturation occurs by separation of portion of nucleus

2 IMPREGNATION OF FEMALE GAMETOCYTE Flagella become free enter and impregnate female gametocytes The resulting cell has power of movement the travelling vermicule or *zygote*

3 PENETRATION OF STOMACH WALL *The 'cyst'* The zygote penetrates mucous membrane settles beneath it requires a definite wall or *sporocyst* and thus forms the oocyst

4 FORMATION OF SPOROZOITES The oocyst grows by division into numerous cell (sporoblasts) and these further divide until finally the oocyst now 50 μ in diameter is full of fine spindle shaped sporozoites, staining blue, with central chromatin nucleus It ruptures and the sporozoites reach the salivary glands of the mosquito and thence on biting, pass into human body, causing infection and commencing human asexual cycle

Duration of cycle For malignant variety twelve days For others, seven to ten days Mosquito in interval is not infective

The Mosquito. Genus *Anopheles* is sole host of malarial parasite numerous species exist in America and Europe especially *A maculipennis* *Culex* the common mosquito in houses, breeds in tanks etc, near dwellings when resting one part or less is elevated above body, a definite distinction from *Anopheles* *Anopheles* breeds in sluggish streams and small pools activity is confined to night

NOTE Female mosquitoes only are blood suckers. males feed solely on vegetable juices Range of flight is very limited, not exceeding half a mile.

Malarial Fever, *continued*

Varieties of Hæmamœbæ.—Man is sole intermediate host of human varieties, other animals being immune Two species occur in birds (1) *Halteridium*—pigeons, etc , (2) *Proteosoma* sparrows, etc , mosquito forming definitive host

MORBID ANATOMY.

Mortality is due to pernicious forms, chronic cachexia, and rarely rupture of spleen common acute malaria is not fatal

PERNICIOUS FORMS (nearly always *P falciparum*) *Spleen* moderately enlarged and very soft *Pigment* present in spleen liver, brain, bone marrow

In cerebral type numerous parasites in small cerebral vessels and in algid type, in intestinal vessels

MALARIAL CACHEXIA —(1) *Anæmia* severe (2) *Spleen* very large—5 to 10 pounds (3) *Liver* usually enlarged (4) *Pigment* in large amounts in spleen liver kidneys and intestines causing a slaty appearance Definite nephritis and cirrhosis of liver may be present

The Blood.—

A. ACUTE FORMS OF MALARIA (1) *Malarial parasites* (2) *Red cells* number reduced Earlier paroxysms may cause large reduction, but effect of individual paroxysm becomes less on repetition *Hæmoglobin* reduced in proportion to red cells (3) *Leucocytes* Leucopenia with relative lymphocytosis Large mononuclears increased (May be leucocytosis during paroxysm) (4) *Pigment* Brown yellowish or black in clumps or various shapes free in blood or in phagocytic leucocytes May contain or be free from iron

B MALARIAL CACHEXIA Changes of a secondary anæmia (1) *Red cells* reduced often 2 000 000 per c mm *Hæmoglobin* reduced and colour index low (2) *Leucocytes* Leucopenia with relative lymphocytosis *Parasites* usually scanty, may need prolonged search Pigment generally slight 'Schüffner's dots' may be present in red cells

Pathogenesis.—Little understood Febrile paroxysm coincides with sporulation and is ascribed to toxins not free Anæmia results from destruction of red cells by parasite, the hæmoglobin being the origin of the pigment.

CLINICAL VARIETIES AND FEATURES.

Can be based upon (*A*) Type of fever (i) Regular intermittent, (ii) Irregular, remittent, (iii) Continuous and pernicious forms (*B*) Type of parasite Both unsatisfactory thus malignant tertian occasionally produces regular paroxysms, and benign tertian and quartan may, though rarely, produce remittent and pernicious forms

CLASSIFICATION. —(1) *Benign tertian fever* (2) *Quartan fever* (3) *Malignant tertian fever* (*a*) Regular intermittent form, (*b*) Irregular and remittent forms, (*c*) Pernicious forms—especially (i) Comatose and cerebral type, (ii) Algid type, (iii) Bilious remittent fever (4) *Malarial cachexia* (5) Latent infections and relapses For blackwater fever and hæmoglobinuria, see p 178

1. Benign Tertian Fever.

Benign tertian and quartan both produce typical 'ague' viz regularly recurrent paroxysms of fever with three stages (1) Cold stage (2) Hot stage (3) Sweating stage *Incubation period* from infection doubtful, experimentally two to fifteen days

Clinical Characters of a Paroxysm. —

PREMONITORY STAGE —Sensations of discomfort for a few hours

COLD STAGE Onset lassitude, headache, often nausea and yawning *Rigor* commences with chill and rapidly becomes extreme *Temperature* rapidly rises to 104 to 106° the maximum of the paroxysm *Skin* cold and blue *Pulse* rapid and weak *Headache* often severe *Vomiting* common *Duration* one quarter to one or even two hours

HOT STAGE Patient becomes very hot, beginning with flushes of heat Face, hands, and skin congested Complains of heat and intense headaches Very thirsty Nausea ceases Temperature begins to fall Pulse full Respiration rapid *Duration* From half to four or even six hours

SWEATING STAGE Perspiration on face, then general may be extreme Feeling of comfort often sleeps

SPLEEN often palpable during paroxysm

HERPES FACIALIS and BRONCHITIS common

VARIATIONS Rigor and cold stage often slight hot stage being most prominent Severity of paroxysms varies greatly.

TOTAL DURATION Usually ten to twelve hours

INTERVAL Usually no symptoms *Length of interval* Single benign tertian infection has cycle of forty-eight hours paroxysm often commences regularly *at same hour* most commonly between midday and midnight Quotidian (daily paroxysm may result from (*a*) double benign tertian (*b*) triple quartan infection (*c*) sometimes in malignant tertian infections

MIXED INFECTIONS of the parasites may occur, producing complex cycles

Course and Progress. *Quinine controls paroxysms readily* Recurrences frequent after long freedom attacks may follow an operation or illness Repeated attacks cause anæmia and chronic malarial cachexia In absence of quinine, paroxysms may cease in a few weeks

Malarial Fever, *continued*

2. Quartan Fever.

Due to *P. malariæ*. Clinically resembles benign tertian fever, but cycle is seventy-two hours. Tendency to relapses.

3. Malignant Tertian or Æstivo-autumnal Fever.

Season. In temperate regions, mainly in summer and autumn. In tropics, in all seasons.

Cycle. May be twenty-four or forty-eight hours; probably variable. Possibly two varieties exist, corresponding to these periods.

Character of fever, symptoms and course tend to be irregular and vary greatly.

a. Regular Intermittent Form. Resembles, in general, the paroxysms in benign tertian and quartan. Note: (1) *Duration* of attack: sixteen to thirty-six hours. *Length of cycle* variable: approximately forty-eight hours. Interval a few hours only. (2) *Cold stage* often very slight: chilly sensations along spine. (3) Temperature rises and falls more slowly.

b. Irregular and Remittent Forms. *Types of Fever:* (a) Continuous fever without paroxysms. (b) Irregular fever with remissions and paroxysms. Possibly due to overlapping of paroxysms.

CLINICAL FEATURES. Very variable. Types are: (i) Weakness; tongue furred; temperature 101° to 103; pulse full; spleen enlarged. Closely resembles enteric fever, but diarrhœa rare. (ii) Paroxysms usually slight; occur irregularly. No definite rigor. Irregular rises of temperature.

Other not infrequent symptoms: jaundice (slight), delirium.

COURSE AND PROGNOSIS. Controlled by quinine with rare exceptions. If untreated: (i) May subside in one to two weeks only in mild cases. (ii) May simulate enteric fever (so called 'typho-malarial fever'). (iii) Anæmia and weakness increase; condition becomes serious. Various pernicious forms may develop.

c. Pernicious Forms.—Common in tropics; rare in cooler districts. Mortality high.

PATHOGENESIS. Localization of parasites in certain sites occurs in some forms. Influence of toxins uncertain.

Note. Benign tertian and quartan fever may assume pernicious forms, but this is rare.

(1) COMATOSE FORM (*Cerebral malaria*). Commonest pernicious type; mortality very high. Parasites very numerous in brain vessels.

TYPES.—(a) Commences with febrile paroxysm; gradual increase of stupor passing into coma, usually of quiet type. Temperature variable; often high, but may be normal. Acute delirium may precede the coma. Terminations

(f) Unconscious twelve to twenty-four hours, then recovery. (g) Frequently fatal. (h) Recovers consciousness, followed by a *second and fatal coma*, common. (i) Hyperpyrexial type. Temperature during paroxysm continues to rise may be manic, then coma and death. Often diagnosed as 'heat stroke'. (j) Sudden coma, resembling apoplexy. Variable temperature, 101° to 103°. Usually previous malaria. Fatal in one to two days. Rare.

ii. ALGID FORM

'ADYNAMIC TYPE' (Due to malarial infection of the supra renals.) *Extreme prostration and weakness*. Pulse feeble. Temperature often subnormal, or slight rise. Respiration rapid. Vomiting common. Feels cold. Urine diminished. *Death* frequent, may be conscious to end.

CHOLERAIC TYPE' Similar, with extreme diarrhœa and vomiting. Parasites numerous in intestinal mucosa and vessels.

iii. BILIOUS REMITTENT FEVER' Predominant symptoms: (a) Jaundice. (b) Vomiting of bile stained fluid. Epigastric pain, hiccough and hematemesis and hemorrhages common. Fatal if untreated.

RARE SEQUELÆ AND COMPLICATIONS

Peripheral neuritis.

Hemiplegia. May occur (*a*) in comatose form, (*b*) at height of ordinary paroxysm.

Aphasia. In comatose form, usually transient.

Conditions of acute ataxia, or of disseminated sclerosis, very rarely.

4. Malarial Cachexia

Occurs with chronic malaria. Characterized by: (1) Anæmia. (2) Enlarged spleen.

SYMPTOMS. (a) Skin of grayish hue. (b) Symptoms of secondary anæmia. (c) Spleen greatly enlarged. (d) Fever, occasional rises.

BLOOD. Parasites scanty. *See* MORBID ANATOMY.

COURSE. Amenable to prolonged treatment.

5. Latent Infections and Relapses.

Paroxysms with parasites in blood may appear many months or even years after possibility of infection; similarly relapses may occur after long latent periods. Surgical operation or ill health may be exciting cause. *Theories* accounting for the latent period include:

ROSS. Small number of parasites persist undergoing normal cycle but insufficient to produce pyrexia until sudden increase occurs. Widely accepted.

SCHAUDINN. 'Parthenogenesis of the macrogametocytes', i.e., these resistant sexual forms linger in spleen, etc., and then finally sporulate, producing asexual spores ('merozoites') which commence recurrent asexual cy

Malarial Fever—Latent Infections, *continued.*

CRAIG AND OTHERS.—A resting stage of the parasite, which lies dormant in spleen, etc., and then renews activity.

Complications. Other diseases may co-exist: *Typhoid fever* (a clinical entity 'typho-malarial fever' does not exist); *pneumonia*; nephritis; dysentery.

DIAGNOSIS.

DIAGNOSIS FROM:—

1. Other tropical fevers, e.g., kala-azar.
2. Enteric fever. Clinically may be impossible.
3. Tuberculosis, with hectic temperature.
4. Severe forms from heat stroke; hæmorrhage, yellow fever.
5. Chronic forms from other causes of large spleen and anæmia.

METHODS OF DIAGNOSIS

1. Presence of malarial parasites.
2. Therapeutic test: *an intermittent fever* resisting quinine is not malaria (Osler).
3. Periodicity of fever (not conclusive). Enlarged spleen.

EXAMINATION OF BLOOD FOR PARASITES.

Note. Examination most valuable shortly *before* a paroxysm is due, and not during height of attack, when sporulation has lately occurred, except for malignant forms. Quinine should be withheld in doubtful cases until blood has been taken, unless urgent.

1. Fresh blood. A drop on a slide under a cover slip ringed with vaseline. Needs experience.
2. Film of blood stained by a Romanowsky method.
3. Ross's thick film method. A drop on a slide: dried, red cells carefully hæmolysed with distilled or tap water, dried, and stained by Romanowsky method. (Rapid and effective if parasites scanty.)

Species of Plasmodium. Malignant tertian is proved by presence of 'crescents', and strongly suggested if 'ring forms' are numerous. (*See also above*, VARIETIES OF PARASITE.)

BLOOD. *See* MORBID ANATOMY.

TREATMENT.

Quinine is a true specific remedy, directly destroying the plasmodium. Action is most marked on spores or 'merozoites', and thus of maximum effect immediately before a paroxysm; but most reliable method is by regular doses. Action on gametes (sexual cells) none or very slight, but none develop if treatment is early.

General Treatment. Rest in bed. Much fluid. Light diet. Bowels open (calomel and salines).

COLD STAGE.—Hot blankets and warmth.

COLLAPSE.—Stimulants, brandy, etc.

HYPERPYREXIA. Cold baths.

GREAT RESTLESSNESS, and in CHOLERAIC FORM. Opium.

Quinine.—

PREPARATIONS.—Best are: (1) Bisulphate, cheap and effective. (2) Bihydrochloride, very soluble and best for injections. (3) 'Euquinine' (ethylcarbonate), tasteless, useful for children, similar dosage.

METHODS OF ADMINISTRATION.—

By Mouth.—Effective except in special circumstances. Administer in solution. Pills, etc., often unabsorbed.

Intramuscular Injections.—When not tolerated orally, and in resistant cases. *Absolute asepsis is essential* of skin, syringes, etc., owing to frequency otherwise of abscesses and tetanus. Dissolve quinine in absolute alcohol and add sterile water or saline (♏xx). Inject into gluteus maximus.

Intravenous Injections.—In urgent cases, especially cerebral and algid forms. Some danger. Use dilute solution, gr. xv in ℥x normal saline, inject slowly. (May be given in 10 c.c., but dilute solution preferable.)

Other Methods.—Subcutaneous, absorption slight owing to formation of coagulum. Rectal injection, absorption slight.

INTOLERANCE.—For cinchonism, give hydrobromic acid. For vomiting, alkalis or tinct. iodi. If it continues, give injections.

PREGNANCY.—not a contra indication.

TERTIAN AND QUARTAN PAROXYSMS. *Dosage.*—By mouth, gr. v t.d.s. for two weeks or up to gr. xx daily. Then two weeks gr. x daily, two weeks gr. v daily, subsequently for three months gr. v twice weekly.

MALIGNANT TERTIAN PAROXYSMS. *Dosage.*—By mouth, gr. x four hourly for three days. Then gr. x. to xx daily for three months. With severe forms gr. x intramuscular per diem until controlled, then by mouth.

PERNICIOUS FORMS. *Dosage.*—Intramuscularly gr. x, repeat in two hours. Subsequently daily by condition, gr. xl to lx may be given in first twenty-four hours by intramuscular injections. Intravenous injections in coma, urgent.

MALARIAL CACHEXIA.—Remove to non-malarious locality. Quinine, if parasites present. Good food, and fresh air. Sanic. iron, and strychnine tonics.

LATENT CASES.—Prolonged treatment with quinine.

Prophylaxis.—Malaria can be brought under control, accomplished by Ross in Ismailia, by Gorgas and others in Panama Canal zone and in Havana.

Prophylactic measures include:

1. Breeding sites of mosquitoes destroyed by drainage, destruction of shallow pools, etc.
2. In large areas, kerosene poured on pools and shallow streams, and banks smeared with insecticide.
3. Destruction of *Anopheles* in houses.
4. Isolation of malarial patients and infected persons, to prevent infection of mosquitoes.

Malarial Fever—Prophylaxis, *continued.*

5. Screening of houses: mosquito nets over beds insufficient.
6. Efficient treatment with quinine: (a) All persons entering a malarial district to take 10 grains daily; (b) Prolonged and thorough treatment of all infected persons, whether symptoms be present or not.

Estimation of Prevalence or Endemic Index of a District.—Many individuals, especially native children, show few symptoms. Prevalence estimated by: (a) 'Parasite rate': percentage in whom parasites are present. (b) Ross's 'spleen rate': rapidly performed and is of sufficient accuracy.

IMMUNITY TO MALARIA. Malaria in negroes appears less serious than in Europeans; possibly due to great frequency of infection in childhood and survival of the fittest.

CHAPTER XXI.

BLACKWATER FEVER.*

(*Malarial Hæmoglobinuria*)

A febrile condition characterized by *hæmoglobinuria*, jaundice, and bilious vomiting, with rigors and frequently diminution or suppression of urine.

Etiology.—The essential change is great hæmolysis of red cells. Two factors are generally accepted. (1) *Malaria*. (2) *Quinine*.

In support of this etiology: (i) Previous malaria and quinine almost invariable. A few apparently authentic cases without quinine are recorded. (ii) Malaria and blackwater fever have similar distribution; although latter is very rare in Roman Campagna. (iii) During and shortly after attack, administration of quinine will often cause reappearance of hæmoglobinuria.

An unknown specific organism is upheld by a few authorities.

Note.—(1) Destruction of red cells is due to a hæmolysin not directly by malarial parasites. (2) Malarial parasites may be absent: when present usually disappear after first day usually, but not invariably, malignant tertian. (3) Onset usually in 2nd and 3rd years of residence in malarial district. (4) Quinine without malaria does not cause hæmoglobinuria.

Morbid Anatomy.—

SPLEEN.—Enlarged and soft. Active phagocytosis present.
LIVER.—Enlarged and soft. Often degenerated.
KIDNEYS.—Tubules contain débris and casts. Epithelium little altered.

Symptoms.—*Prodromal.*—Ordinary attacks of malaria. *Onset.*—Commences with rigors, often recurrent, several hours.

* Blackwater fever is placed after malaria for convenience. It is probable, but not proved, that it is malarial in origin.

HAEMOGLOBINURIA.—Urgent desire to micturate after a rigor; dark urine passed. Duration of dark urine few hours to one day: rarely exceeds two days.

TEMPERATURE.—103° to 105°: irregular. Falls as urine clears.

BILIOUS VOMITING.—Much retching and epigastric pain.

ICTERUS.—Within 24 hours of onset; becomes intense.

GENERAL SYMPTOMS.—Restless. Pain in loins. Great thirst. Exhaustion. Liver and spleen enlarge.

PROGRESS.—(*a*) *Recovery.* Urine clears, and temperature falls: sweats and then symptoms pass away. Post-hæmoglobinuric fever: occasional pyrexia frequent for several weeks. (*b*) Symptoms increase. Restlessness, rigors, high temperature, *Thirst extreme.* Hiccough serious. Urine diminished: final anuria common: fatal termination.

DEATH from: (i) *Cardiac failure*—great exhaustion; (ii) *Anuria*; (iii) *Hyperpyrexia.* Coma or delirium common.

MORTALITY.—About 25 per cent.

RELAPSES and RECURRENCES not uncommon.

Urine.—In early stages, amount increased and micturition frequent. On standing, separates into two layers: (*i*) Clear and dark. Gives spectroscope of oxy- and methæmoglobin. (*ii*) Large, dark sediment, consisting of much débris and casts. Albumin present: almost solid on boiling. Bile rarely present.

Blood.—Red cells reduced to 1,000,000. Hæmoglobin 20 per cent. Colour index normal. Red cells present are practically unchanged. During attack, polynuclears form 90 per cent of leucocytes. Later mononuclears high, with leucopenia.

Diagnosis.—From (1) Yellow fever; (2) 'Bilious remittent fever' of malaria. In blackwater fever, rigor, pyrexia, and hæmoglobinuria occur together at onset.

Treatment.—

REST IN BED.—Absolute, as syncope may occur.

PROMOTE SECRETION OF URINE.—Large quantities of bland fluid. If prevented by vomiting, give rectal or subcutaneous saline injection.

CARDIAC STIMULANTS.—e.g., ether, alcohol, camphor.

VOMITING.—Ice to suck, champagne.

SUPPRESSION OF URINE.—Fomentations to loins. Avoid diuretics.

QUININE.—*Not to be given during attack*, except that it may be given on first day if parasites numerous.

After convalescence, patient should leave malarial districts.

AMŒBIC DYSENTERY.

(*See* DYSENTERY, p. 92.)

CHAPTER XXII

TRYPANOSOMIASIS. LEISHMANIASIS.

TRYPANOSOMIASIS.

(*Sleeping Sickness*)

An infection by *Trypanosoma gambiense* producing a long continued malaise and pyrexia and finally a prolonged lethargic condition

History.—Progress in discovery of trypanosomes has been

1 Non pathological and in animals Gruby, 1843 in frogs, later others found in birds and fishes Lewis, 1878, in rats (*T lewisi*)
2 Pathological and in animals 1880, Evans, in Surra disease of horses (*T evansi*) 1895, Bruce, in Nagana, tsetse fly disease in S Africa (*T brucei*)
3 Pathological and in man 1901 Dutton pathological nature not recognized, 1903, Castellani followed by Bruce and Nabarro in blood and cerebrospinal fluid of 'sleeping sickness' (*T gambiense*)

Note Following section refers to human trypanosomiasis or 'sleeping sickness'

Distribution. Gambia Sierra Leone, and West Africa were original districts From Congo spread by opening of intercourse to Uganda causing enormous mortality Rhodesia recently became infected (*T rhodesiense*) Both natives and Europeans susceptible

Mode of Infection. Infection is conveyed only by tsetse fly species

1 *Glossina palpalis* Breeds on lake and river bank in thick forest Hence these localities are specially liable In addition to many *big game* (and especially crocodiles) they form a reservoir without production of symptoms In Gambia Uganda, etc, no other tsetse fly conveys infection
2 *Glossina morsitans* Prevalent in Rhodesia Can convey infection Breeds in any locality

The Trypanosome.

MORPHOLOGY —*Protozoa* subclass *Flagellata* Stained by Leishman's or similar methods possess following characteristics

1 Unicellular roughly fusiform shape length about 20 μ breadth 1 5 to 3 μ (variable) protoplasm stains blue and contains two nuclei
2 *Macro* or *trophonucleus* near middle stains purple red
3 *Micro* or *kinetonucleus* near posterior end small stains intense deep purple red
4. *Undulating membrane* commences near kinetonucleus margin stains purple runs entire length, and is continuous with
5 *Flagellum* at opposite end to kinetonucleus Since progression is usually in direction of flagellum, this is regarded as *anterior* end.

In fresh blood is actively motile, by movements of undulating membrane and flagellum

CULTIVATION In Novy and McNeal's medium (broth with twice volume of defibrinated rabbit's blood)

LIFE CYCLE Two phases (1) In blood of vertebrate host (man or big game) (2) In gut of blood sucking invertebrate host (*Glossina*) Life cycle and possibility of sexual stages at present incompletely known

In *Glossina palpalis* Trypanosomes enter with sucked blood reach gut None in proboscis after forty eight hours After five to seven days none present in gut Subsequent stages unknown but reappear in gut (in small proportion of flies) in large numbers after eighteen to twenty five days whence reach salivary glands in position for infection Thus until about the thirty second day fly does not convey infection by bite

[*Leptomonas* in rat flea and also in cultures passes through spherical stages resembling *Leishmania* Multiplication of trypanosome can occur by unipolar fission longitudinally commencing with kinetonucleus flagellum [illegible] thin [illegible] with numerous fine trypanosomes united by fine filament before final separation Before division both greatly increases In all trypanosomes forms vary greatly in breadth but it is unknown whether these are sexual forms

Prophylaxis. Chief factor to be considered to prevent spread

HUMAN HOSTS Prevention of movement of natives from infected to uninfected districts Native may carry trypanosomes in blood with or [illegible] but [illegible] *Glossina* [illegible] when [illegible] of all [illegible] natives after examination

TSETSE FLY *Glossina* breed in thick forest on water banks these can be cleared [illegible] breeds widely

BIG GAME Not is reserves Destruction under [illegible]

Morbid Anatomy. Nothing distinctive except in brain

BRAIN Fluid increased congestions [illegible] Often a terminal purulent meningitis

HISTOLOGY OF CENTRAL NERVOUS SYSTEM A characteristic meningo encephalomyelitis most marked at base of brain and medulla (Mott) great infiltration of mononuclear leucocytes in perivascular spaces sufficient to interfere by pressure with circulation and hence with nutrition of nerve cells

LYMPHATIC GLANDS Enlarged in early stages (pea to bean)

Symptoms. Long latent period Very insidious onset Three stages with *gradual transition*

STAGE I *Trypanosome fever* Trypanosomes in blood but not in cerebrospinal fluid, also present in gland puncture Lymphatic glands palpable Pulse often rapid some mental dullness

Trypanosomiasis Symptoms, *continued*

Spleen may be large Duration three months to three years Recovery possible

STAGE 2 — *Stage of tremors* Expression vacant but, with effort, intelligence good Speech slow and weak Gait shuffling Tremors tongue, hands, and feet Fever irregular, 101° to 102° in evening Trypanosomes in blood and cerebrospinal fluid

STAGE 3 *Stage of lethargy* Apathetic condition eyes open not truly 'sleeping' Unable to stand or speak Wasting results only from inefficient feeding Temperature very low 95° to 94° Duration about eighteen months Death in lethargy or frequently with terminal purulent meningitis

Prognosis. Invariably fatal if any symptoms as above develop Trypanosomes in blood may disappear without symptoms occurring very rarely or occasionally persist in natives with febrile attacks only

Diagnosis. By presence of trypanosomes Earliest by puncture of lymph glands trypanosomes present early in numbers In blood, scanty necessary to centrifugalize to concentrate blood In later stages in cerebrospinal fluid *Blood* increased percentage of lymphocytes and large mononucleats

Treatment. —Arsenic and antimony have most effect on reduction of trypanosomes Intravenous injections of tartar emetic (gr ss to iss) can be given daily in series of ten or twice weekly for prolonged periods recovery has been recorded Combined with occasional injections of atoxyl (gr iij to v) probably most effectual No cure after development of symptoms nor experimentally in Ngana (Atoxyl may cause optic neuritis)

LEISHMANIASIS.

(*Kala Azar Tropical Sore*)

A group of diseases caused by flagellate protozoa of the genus *Leishmania* Three forms known (1) Indian kala azar (2) Infantile kala azar, (3) Tropical sore Parasites while closely allied are not identical

Indian Kala-Azar.

DISTRIBUTION OF DISEASE Widely throughout Asia, also in Soudan Europeans not exempt

CHARACTERISTIC SYMPTOMS (1) Enlargement of spleen (2) Irregular pyrexia many months, (3) Progressive anæmia and cachexia Leucopenia is marked Leishman, 1900 discovered bodies in spleen smears studied also by Donovan organism known as *Leishmania donovani* ('Leishman Donovan bodies')

MORPHOLOGY In smears from spleen with Romanowsky stain small 'cockle shaped' bodies, about 2·5 to 3·5 μ protoplasm pink or blue, and contains (a) small nucleus near periphery

staining intense red; (b) larger nucleus nearer centre, staining less deeply; also usually vacuolas. In smears lie free, but in sections are mainly intracellular in larger endothelial cells

CULTIVATION Cultivated in original spleen juice, a motile flagellate organism develops. Resembles to some extent trypano somata, but no undulating membrane Life cycle incompletely known

OCCURRENCE IN BODY —In spleen, liver, and bone marrow. Also in blood in *very small numbers.*

METHOD OF INFECTION —Probably by bed-bug, in which experiments have shown flagellate forms. Cannot be conveyed to animals

DIAGNOSIS By symptoms, combined with absence of malarial protozoa, confirmed by spleen puncture (fine hypodermic needle)

MORTALITY - Formerly 80 per cent. Now falling rapidly under tartar emetic

Infantile Kala-Azar.—

DISTRIBUTION - Shores of the Mediterranean. Widespread Is 'infantile splenic anæmia'.

CHARACTERISTICS (1) Confined to children, ages two to five years (2) Enlarged spleen, progressive anæmia (3) Dogs, monkeys, rabbits, and other animals are susceptible Dogs in endemic area harbour similar parasites infection probably by dog fleas or human fleas biting dogs (4) Parasite present in spleen, liver and bone marrow Morphologically resembles *L donovani* differs in effects on dogs, etc known as *L infantum* Discovered by Nicolle in Tunis

Tropical Sore.

Also known as Baghdad boil, Delhi sore tropical ulcer, etc Chronic ulcerating lesions In discharge are bodies resembling *L donovani* (*L. tropica* J H Wright) Mode of infection unknown. Incubation period, two months Duration, about one year

LOCAL TREATMENT Mild antiseptics Vaseline with methylene blue or other dye Good results reported from X rays.

General Treatment of Leishmaniases. Antimony intravenous injections of tartar emetic Good results reported in all forms For technique, *see* BILHARZIASIS (p 185)

Section I.—Specific Infectious Diseases, *continued*

D. DISEASES DUE TO METAZOAN PARASITES.

CHAPTER XXIII

TREMATODE OR FLUKE INFECTIONS.

(*Distomiasis*)

Four principal groups of fluke infections occur in man. Distribution is confined practically to tropical and subtropical regions, but *Bilharzia* persists in persons returning to cooler climates if untreated.

1. **Pulmonary Distomiasis.** *Endemic Hæmoptysis.*—
A fluke, *Paragonimus westermanni*, size 8 to 10 mm. long by 4 to 8 broad, present in lungs. Mainly in China and Japan.
SYMPTOMS. (a) Hæmoptysis, slight or occasionally severe. (b) Cough. Condition often suggests tuberculosis. (c) Ova in sputum in large numbers, oval, about 100 × 50 μ. Cerebral abscess may occur. No specific treatment.

2. **Hepatic Distomiasis.**—Many varieties of flukes. Cirrhosis of liver occurs, with ascites, etc. In some forms splenomegaly.

3. **Intestinal Distomiasis.**—Intestinal flukes.

4. **Blood Flukes.**—*Bilharziasis.*

BILHARZIASIS.

(*Endemic Hæmaturia.*)

A chronic infection by a species of blood fluke, symptoms being produced by passage of ova from bladder or rectum.

Distribution. Widespread throughout North and South Africa, parts of India, and other countries. In Egypt, probably half the native population affected. Bilharz, 1851, discovered parasite.

The Parasite. Sexes distinct. (1) *Male*, length 11 to 15 mm. by 1 mm. broad, sides curved to form an unclosed cylinder (the characteristic *gynæcophoric canal*), has two suckers, body covered by spinous prominences. (2) *Female*, much longer but filiform. When young, sexes are separate, but at maturity female enters gynæcophoric canal of male, projecting at each end owing to greater length.

TYPES. Two types occur, with following differences:—

(1) *Schistosoma hæmatobium* or *Bilharzia hæmatobia*. (i) Ovum has a terminal spine. (ii) Ova penetrate bladder, causing hæmaturia, and appear in urine. (iii) Miracidium set free from ovum enters a snail, *Bullinus*.

(2) *Schistosoma mansoni*.—(i) Ovum has a lateral spine. (ii) Ova penetrate rectum, causing blood in stools; (iii) Miracidium from ovum enters a snail, *Planorbis boissyi*.

LIFE CYCLE. Leiper, 1916, determined extravertebrate stages

1. Parasites inhabit portal vein of man, grow to maturity female enters gynæcophoric canal of male, together migrate to smaller veins of bladder or rectum: ova deposited
2. Ova traverse tissues and reach bladder or rectum, an embryo is now present in ovum On reaching water an active embryo, covered with cilia, escapes (miracidium).
3. Miracidium penetrates a fresh water snail, reaches liver, forms sporocysts in enormous numbers, whence bifid tailed cercariæ escape into the water
4. Cercaria penetrates skin of man (or animal), shedding its tail Thence reaches portal vein and attains maturity in about six weeks Can pass through mucous membrane of mouth

Morbid Anatomy. Ova passing through tissues act as foreign bodies, causing irritation and fibrosis On bladder wall form prominences, often near trigone from collections of ova, many becoming calcified Subsequently, cystitis and papillomatous growths of bladder and ulceration occur, and suppuration round bladder occasionally malignant tumours Equivalent changes in rectum and in women vaginitis

Ova also in kidney and other tissue

Symptoms.

INITIAL STAGE Four to ten weeks after infection Constitutional disturbances fever abdominal pain cough diarrhœa urticaria, enlargement of spleen and liver Interval of months or years before pelvic viscera affected

PELVIC VISCERA

a BLADDER *Bilharzia hæmatobia* (1) Hæmaturia especially at end of micturition, (2) Aching in perineum (3) Chronic cystitis (4) Ova in urine, with terminal spine

Complications Urinary fistulæ from perirurethral and perineal abscesses very common Calculi in bladder and kidney

b RECTUM *S. mansoni* (1) Blood and mucus in stools with tenesmus, (2) Chronic ulcerative proctitis (3) Ova in stools with lateral spine

Complications Prolapse common Papillomatous growths Vaginitis

BLOOD Eosinophilia, 5 to 10 per cent Anæmia rarely severe

DURATION Two years or often longer

PROGRESS Condition becomes a chronic cystitis, cause being not necessarily fatal, but if neglected ordinary sequelæ of sepsis, etc, follow With suitable treatment health is maintained Death from intercurrent disease or neglect of bladder

Diagnosis. Ova are characteristic

Treatment. Intravenous injections of tartar emetic (Christopherson) specific cure Dissolve in 2 to 6 ounces of sterile normal saline prepare within few hours of use. Dosage commence with gr. ss, increasing to gr. ij Injections alternate days Total about 20 to 30 gr Numerous cures. In cystitis. For rectum, local sedatives.

CHAPTER XXIV.

DISEASES CAUSED BY CESTODES.

(*Tæniasis*)

1. INTESTINAL TAPE-WORMS.

Tæniæ or 'tape-worms' are flat, with a varying number of segments. Adult parasites, the sexual stage, live in the small intestine; the larvæ in the muscles and solid organs. The most important varieties in man are: (1) *Tænia solium*, pork tape-worm; (2) *Tænia saginata* or *mediocanellata*, beef tape-worm; (3) *Tænia echinococcus*, larval. Less frequent are: (4) *Tænia cucumerina*, common in dogs and occasionally in children. Intermediate host: dog-fleas and lice. Head, 4 suckers and hooklets. (5) *Tænia nana*, dwarf tape-worm. Host: rats, mice, man. Infection from droppings of rats and mice. Length 5 to 45 mm.; about 200 segments. Not uncommon in United States; rare elsewhere. Large numbers may be present. (6) *Dibothriocephalus latus*. In Finland, Baltic, and Switzerland; rare elsewhere, imported to United States. Host: man, dog. Intermediate host: pike and other fish. Can produce blood changes identical with pernicious anæmia, but curable on discharge of worm.

The principal characteristics are given in the table on p. 187.

Tænia Solium.—Life cycle. The uterus contains numerous ova. If ova are ingested into stomach of pig, embryo becomes free, penetrates wall, reaches muscles (also brain and liver), develops into larval form or cysticercus cellulosæ, constituting 'measled pork.' Cysticerci are known as measles or bladder worms. Frequent sites: tongue, muscles of mastication, shoulders, neck, diaphragm. In man, eating such pork, larvæ develop into adult worms. Rarely larval forms occur in man (*see* CYSTICERCUS CELLULOSÆ) *with serious results.*

Tænia Saginata. Life cycle resembles *T. solium*, but cattle form intermediate host. Cysticerci most common in muscles of jaw. Larval forms never occur in man.

Tænia Echinococcus.—*Only larval forms* occur in man (*see* T. ECHINOCOCCUS, p. 189). The following description of symptoms, etc., does not refer to this variety.

Symptoms.—Occur at all ages. May be no symptoms.

IN CHILDREN.—Appetite often ravenous or capricious combined with wasting. May be vague pains, itching of nose, anal pruritus. Convulsions, habit spasms, etc., frequently ascribed to this cause, but connection often doubtful or indirect.

IN ADULTS.—Knowledge of infection often causes 'depression' especially in nervous women.

BLOOD. Eosinophilia present usually.

INTESTINAL TAPE-WORMS.

CHARACTERISTICS	*T. solium*	*T. saginata*	*T. echinococcus*	*Dibothriocephalus latus.*
Distribution	Widespread in Germany, in England and America less common	Widespread; commonest tyT	Australia and Iceland very common: but widespread	Finland, Switzerland; rare elsewhere
Host (adult worms; in intestines)	Man only	Man only	Dog: also wolf and jackal (never in man)	Man, dog
Intermediate host (larval forms; in muscles and solid organs)	Pig: man occasionally	Cattle: never in man	Hog, sheep, ox, and man	Pike and other fish
Length	6 to 12 feet	15 to 20 feet	⅓ inch	25 to 30 feet
Head	Small pin's-head, 4 suckers, rostellum with hooklets	Larger than *T. solium*; 2 mm square: 4 suckers, no hooklets	4 suckers; double row hooklets (hooklets barbed)	2 lateral grooves; no hooklets
Proglottides — Number	Many hundreds	17 8 mm. Many hundreds	4 including head (rarely 3 or 5)	Many hundreds
Proglottides — Size	10 × 7 mm.		—	10 × 2 mm.
Proglottides — Shape	Elongated	Elongated	Elongated	Broad and short
Sexual pore	Lateral	Lateral	—	Central
Uterus	Coarsely branched	Very finely branched	Only terminal segment mature	Rosette: in centre of proglottis
Ova	Nearly spherical; thick shell, contains embryo, (not 1st visible hooklets)	As *T. solium* (differences slight); no hooklets	—	—

Intestinal Tape worms, *continued*

Diagnosis.—Proglottides are pathognomonic. Ova in stools often distinctive.

Prophylaxis. (1) Inspection of meat. Cysticerci in beef die in 3 weeks, but in pork live longer. (2) Sufficient cooking of meat. (3) Destruction by burning of all tape worm segments passed in stools. Infected individuals should guard against auto reinfection, especially with *T. solium*, in which case cysticerci may develop.

Treatment. Must be thorough with adequate preparation. In bed three days on fluid diet; bowels must be opened freely.

First evening. Castor oil.

Second and third days. In morning saline (magnesium sulphate); in evening cascara.

Fourth day (no food until treatment completed).

8 a.m. Liquid extract of male fern, one drachm.

℞ Ext. Filicis Liq. ʒj. Mist. Amyg. Dulc. ad ℥j.
Pulv. Tragacanth. Co. ʒss.

9 a.m. Repeat the draught.

11 a.m. Castor oil or full dose of saline.

12 n. Enema if bowels not opened.

Filix mas is extremely unpleasant; patient must keep absolutely at rest and resist vomiting. Alternative methods are: (1) Capsules containing ♏xv each. (2) Oil of filmaron ʒj, a compound of castor oil and an active principle of male fern; aperient subsequently unnecessary.

Motions after male fern to be passed into warm water, and *the head must be searched for.* If passed tape worm cannot grow again, but if retained will re-form.

If filix mas fails try *pelletierine tannate* gr. vj (active principle of pomegranate bark); add few grains of tannic acid; purge an hour later.

2. CYSTICERCUS CELLULOSÆ.

The presence in man of the larval form of *Tænia solium* (pork tape worm) occurs rarely. The pig is the usual intermediate host for cysticerci. Man occasionally acts as such if ova enter the stomach.

Cysticercus Cellulosæ. Elliptical shape, about ⅓ mm. by ½ mm. (one third of an inch); semi-transparent (hence called bladder worms). Where pressure is slight, e.g., in ventricles of brain, may be larger.

Mode of Infection. (1) Proglottides reach stomach by wandering or result of vomiting; or (2) Ova are ingested from presence on fingers (auto reinfection).

Distribution. Sites are: (*a*) Subcutaneous and in muscle, usual site; (*b*) Central nervous system, occasionally; (*c*) Eye, rarely.

Symptoms. Depend on site and number of cysticerci.

SUBCUTANEOUS AND MUSCULAR. Usually no definite symptoms. If numerous, rarely severe pains. Cysticerci palpable as small, subcutaneous, often painful nodules.

CENTRAL NERVOUS SYSTEM.—In brain produce very varied pressure symptoms headache, various paralyses

EYE May be present in vitreous humour

Diagnosis. By removal of subcutaneous nodules, or very rarely by presence in eye

Treatment. No special treatment.

3. TÆNIA ECHINOCOCCUS: HYDATID CYSTS.

Infection in man by larval forms of *Tænia echinococcus* The characteristic of these larval forms (or hydatid cysts) is the power of multiplication The adult worm never occurs in man

Tænia Echinococcus. For characters see table p. 157)

Hydatid Cysts

DEVELOPMENT The terminal segment of the adult worm is discharged in dog's fæces and ova reach stomach of intermediate host From ovum six hooked embryo (oncosphere) escapes penetrates stomach and reaches various sites liver lungs etc Hooklets are lost and cyst forms

HISTOLOGY OF CYST WALL Two layers (1) External laminated chitinous layer ectocyst (2) Internal parenchymatous layer endocyst Surrounding layer of fibrous tissue forms from host

DEVELOPMENT IN CYST From the endocyst outgrowth form which bud into

1 SECONDARY CYSTS Exactly resembling primary cyst They may become free Also tertiary cysts may similarly develop the whole attaining enormous size

OTHERS Immature heads of *Echinococcus* characterized by sucker and hooklets In at time of birth of host develops into adult tænia

Daughter cysts may be (a) Endogenous usually in animals buds in man developing within parent cyst (b) Exogenous usually in animals buds prolifer cyst wall and develop externally never very large

CONTENTS OF HYDATID CYSTS Clear fluid no albumin (unless repeatedly tapped) specific gravity 1005 to 1010 contains chlorides *Characteristics* (1) Daughter cysts (2) Scolices, as above (3) Barbed hooklets Cysts are often sterile and may contain neither scolices nor hooklets

TERMINATION OF CYSTS (1) Death of parasites, followed by inspissation and calcification Characteristic wall and hooklets may be present Not uncommon (2) Rupture (3) Suppuration The last two are serious

Principal Situations in Human Body. (1) Liver most frequent, (2) Lungs and pleura Less commonly kidneys, nervous system omentum, stomach No site is immune

Hydatid Cyst of Liver.

Symptoms.—None when small. When large, may be dragging pain, or tumour in abdomen; or cough, depending on direction o enlargement. General health unaffected unless *complications* occur, viz :—

RUPTURE OF CYST.—Spontaneous or result of strain. Patient often conscious of 'something giving way'. Urticaria common Directions: (i) Stomach and intestines, most frequent, may discharge for weeks, recovery or death from suppuration (ii) Lungs; fragments of cysts coughed up. Often fatal from suppuration, hæmorrhage, gangrene of lung, suffocation. (iii) Peritoneum; usually fatal peritonitis Other directions may be: bile-ducts, extreme jaundice; pericardium, vena cave

SUPPURATION.—With or without rupture. Symptoms of sepsis rigors, sweats, pyrexia.

Physical Signs.—Depend on position of cyst Most common in right lobe Great enlargement of liver. (a) Downwards, resembling tumour of liver and appearing in epigastrium or hypochondria: especially if cyst on anterior surface or in left lobe. (b) Upwards by compressing lung, closely resembles pleural effusion: heart may be displaced especially with cysts on posterior surface and in right lobe

PALPATION.—*Elastic* sensation if cyst is large, occasionally with fluctuation. '*Hydatid thrill*': On sharp pressure with fingers a thrill may be momentarily felt, like 'quivering jelly', ascribed to impact of daughter cysts rarely obtained

Diagnosis.—

1. CLINICAL Great enlargement of liver, persistent, but associated with *good health* Physical signs, elasticity, fluctuation, thrill, and painlessness
2. CYST FLUID. After aspiration (1) Scolices (2) Barbed hooklets Either distinctive, but both may be absent if cyst sterile. Urticaria and toxic symptoms may follow aspiration
3. BLOOD.—Eosinophilia
4. SERUM DIAGNOSIS. Results uncertain, best method by deviation of complement, precipitin test less reliable

Differential Diagnosis.—

CARCINOMA OF LIVER. Often difficult, except by absence of wasting and good general condition

PLEURAL EFFUSION. -May be impossible clinically: differentiated by puncture fluid

HYDRONEPHROSIS Catheterization of ureter may be necessary

DILATED GALL-BLADDER. Is usually mobile and the shape distinctive.

SYPHILITIC LIVER.—No fluctuation

PANCREATIC AND SIMILAR CYSTS.

Hydatid Cyst of Lung.

Most frequent site next to liver Symptoms result from effect on the lung tissue, pressure on bronchi, etc, which may produce (i) Bronchitis, occasionally fœtid bronchitis, bronchiectasis, gangrene (ii) Compression of lung with signs of consolidation (iii) Hæmoptysis (iv) Cavitation (v) Pleurisy and empyema Condition often suggests phthisis Prognosis serious Hooklets may be present in sputum X ray of thorax often decisive, shadow of cyst has sharp regular curved outline

Hydatid Cyst in Pleura.

Less common Simulates pleural effusion General health good until complications occur viz, (1) rupture into lung or occasionally external (2) suppuration when prognosis is serious

Hydatid Cyst of Kidney.

Not common May resemble hydronephrosis Rupture into pelvis and passage of contents in urine, or into peritoneum and tissues

Hydatid Cyst of Brain.

Rare. Symptoms of tumour, usually cerebral

Treatment of Hydatid Cyst. Treatment surgical Cyst should be opened and evacuated If suppurating treatment as for abscess Small cysts occasionally can be excised entire

Aspiration except for diagnosis is not advisable owing to frequent failure and to risk of suppuration extension and toxic effect

CHAPTER XXV

DISEASES CAUSED BY NEMATODES.

1. ASCARIASIS

Ascaris Lumbricoides. (*Round Worm*)

Parasite. General resemblance to the earth worm Cylindrical, pointed both ends yellowish colour transverse striations, four longitudinal bands Male length 9 to 10 inches Female length 8 to 16 inches

OVA Oval very thick capsule no sign of embryo numerous in fæces stained brown by bile

LIFE CYCLE No intermediate host After ingestion of ova embryos hatch in upper portion of small intestine, penetrate mucous membrane enter blood stream and reach liver After a few days embryos enter hepatic veins pass through the heart to the lungs escape into the bronchi, pass up trachea and down oesophagus to stomach and intestines, where they reach maturity

Ascariasis—the Parasite, *continued.*

one month after ingestion (Low), compare ANKYLOSTOMIASIS p. 196.

MODE OF INFECTION. By water. By vegetables supplied with infected water. By auto infection.

NUMBER. Often one or two. May be very numerous.

Symptoms.—Often none. In children, especially if nervous, may be various vague symptoms of disturbed digestion, irritability, nose picking, teeth grinding. Cough and perhaps bronchopneumonia probably may result from migrations of embryos.

Wanderings of adult worms often extensive: into bile ducts, producing jaundice; into appendix; into stomach, subsequently being vomited and withdrawn by subject from pharynx. Rarer situations: perforation of intestine and peritonitis; pancreatic duct and fatal pancreatitis. Few possible sites have escaped.

EOSINOPHILIA may occur to moderate extent, but often absent.

Treatment.—Give castor oil at night. Next morning santonin with an aperient, e.g., for a child one to two years old:

℞ Santonin gr. j–ij. Calomel gr. ss.
Pulv. Scammonii gr. ij.
Ft. pulv.

At midday give a saline purgative. Repeat next day. Warn parents of effects of santonin, viz., urine coloured green or red if alkaline; blue vision followed by yellow; may be vertigo. For adult, santonin gr. v., with calomel and saline purgative.

✓ Oxyuris Vermicularis. (*Thread Worm.*)

Parasite.—Male: length 4 mm., tail coiled in spiral. Females 10 mm., tail long and pointed. In fæces often in large numbers, resemble short pieces of thread, moving lowly.

Modes of Infection. Occurs through water or infected vegetable. After ingestion of ova, worms mature in small intestine, then migrate to cæcum, where majority remain and ova are discharged. Some go to rectum, pass through anus, especially during warmth in bed, and cause great irritation. Resultant scratching leaves ova on fingers and reinfection follows.

Symptoms. Mainly in children. Often previously in unhealthy condition, with disturbed digestion and excretion of mucus. Symptoms ascribed to infection: irritability, itching of anus and perineum, insomnia, picking nose, may become hysterical. EOSINOPHILIA. Occasionally present to slight degree.

Treatment.—Treat any disturbance of digestion, e.g., diminish sugar and carbohydrates; give aperients (hydrarg. c. creta). *Treatment of Infection.* Nightly hot rectal washes, once weekly a simple soap and water enema, followed after return by enema of infusion of quassia, 6 to 10 ounces (for a child), hips to be raised and enema retained as long as possible (keep quiet and tie thighs together). For itching of anus, apply ung. gall. c. opio or

carbolized vaseline To prevent auto infection cover child's buttocks at night with drawers or tie nightdress below feet. Anthelmintics by mouth, santonin and male fern, may be tried, as for ascaris and tænia

2. TRICHINIASIS.*

Infection of the human being by *Trichinella spiralis* results in a stage of gastric irritation during the development of the adult worm in the intestine, and a more characteristic stage of myositis due to the migration of the embryos to the voluntary muscles.

Parasite.—

1 ADULT FORM Both sexes are cylindrical, the oral end being pointed Male length about 1 5 mm two projections from posterior end resembling the jaws of a pair of pincers hold the female in coitus Female length 3 to 4 mm Characteristic the œsophagus is lined by a *single layer of large cells*, readily recognized at the anterior portion of the parasite, and known as the *cell body*'

2 EMBRYOS Minute organisms

3 LARVAL FORM or muscle trichinella Oval laminated capsule, length about 0 6 to 1 mm contains a distinct coiled worm with pointed head and rounded posterior end In early stages capsule translucent subsequently impregnation with lime salts occurs and then it is easily visible with a hand lens May be two, and rarely three or four worms in single capsule

Mode of Infection. In man by eating trichinous pork No intermediate host is necessary and thus among hogs in large herds probably spread by feeding on offal of other infected animals In hog symptoms are slight even with large infections also calcification of capsule is less common and cysts more difficult to recognize Rats may be true hosts of the parasite

Muscle trichinellæ are resistant to heat but destroyed at boiling point rendered by thorough cooking of pork but at temperature of a joint temperature may be insufficient

Geographical distribution is universal but human infection is rare except in North Germany where raw ham is consumed

Tends to occur in small outbreaks, but isolated cases are not infrequent

Cycle in Human Body and Mode of Spread.—

1 On ingestion of muscle trichinellæ capsule is digested and larval trichinellæ enter the duodenum and jejunum

2 By the third day the adult worm is fully grown and sexually mature

3 By the sixth to seventh day, embryos are fully developed The

* The terminology has become confused The original name *Trichina spiralis*, given to the parasite was not admissible, as *Trichina* was previously in use, hence it was altered to *Trichinella spiralis* The clinical condition is often referred to as 'Trichinosis', but more correctly is 'Trichiniasis', or most correctly 'Trichinellosis'.

Trichiniasis—Parasite, *continued*

adult female is ovo viviparous, discharging free embryos in large numbers from the uterus; dies after discharging embryos for five or six weeks, many hundreds

4 *Fate of the adult worms* Male dies after copulation

5. *Spread of embryos* The female penetrates intestinal wall, and discharges embryos into lymph spaces, whence they enter veins reach intermuscular connective tissue, and finally enter *voluntary muscle fibres* In early stages embryos have been found in the blood, also numbers have been found in peritoneal and other serous sacs

6 *Embryos in the muscle* The embryo coils, becomes less active, and in two weeks from ingestion of meat definite 'muscle trichinellæ' are present A local myositis results and an oval capsule forms, probably from the muscle The formation of the capsule takes about six weeks If fresh muscle be teased on a warm slide, embryo may be seen to move can remain alive many years but undergoes no further development

Calcification of capsule in man occurs in four to five months, kills embryo, and also renders capsule visible In hog, calcification less common

7 *Muscles affected* Most frequent are the diaphragm intercostals, muscles of neck and eyes and larger voluntary muscles especially near tendinous insertions of voluntary muscles, in man biceps and *gastrocnemius* especially liable

Symptoms.—

The severity of symptoms depends on extent of infection and also on number of embryos developed in small intestine May be very slight, or merely *rheumatic pain* When severe following stages are definite

STAGE OF GASTRO INTESTINAL IRRITATION Corresponds to development of adult worms in small intestine sexual activity and possibly penetration of gut by females *Onset* may be within twenty four hours usually two to three days after ingestion *Abdominal pain, vomiting,* and often diarrhœa

May be absent, or of cholera severity with muscular cramps intensity is no guide to subsequent stage as vomiting and diarrhœa may discharge many adult worms before embryos are free

STAGE OF MYOSITIS Corresponds to migration of embryos and capsulation in muscles *Onset* 7th to 14th day, usually 9th or 10th

(a) *Fever* 102° to 104°, remittent or intermittent (b) *Myositis* Muscles swell, become hard very tender, and all movements painful. Position in bed limbs semiflexed to relax muscles, most typical being *flexed forearm* owing to great *infection of biceps* Other special muscles commonly affected are (a) Diaphragm (cough and respiratory troubles, may be extreme dyspnœa), (b) Muscles of mastication and larynx (aphonia), (c) Muscles of the eye; (d) Gastrocnemius.

(3) *Œdema*, important sign: (*a*) In face. early transient œdema about 8th day. (*b*) In 4th or 5th week: œdema, often extreme, of face, limbs, and entire body (genitals may escape) Albuminuria is rare (4) *Eosinophilia*. Extreme, total leucocytes 20,000 to 30,000 per c mm, and eosinophils may be 50 per cent

VARIOUS SYMPTOMS –

TYPHOIDAL STATE develops with intense infections, resembles severe typhoid delirium, dry tongue, tremors, rapid pulse

EMACIATION and ANÆMIA severe in prolonged cases

Occasionally: pneumonia, pleurisy, sweats, urticaria, and boils.

Duration.—Depends on extent of infection Mild cases recover in two weeks *Severe cases* convalescent in six to eight weeks – sometimes termed 'third stage of subsidence' Many months of weakness follow

Prognosis.—Best in children, and with much early diarrhœa resulting in excretion of adult worms

MORTALITY has varied greatly in different outbreaks depends on degree of infection of flesh at fault varies from 1 to 30 per cent, but usually 25 per cent *Death* usually occurs in 4th to 5th week while myositis severe From (1) Weakness of diaphragm and intercostals, and extreme dyspnœa, (2) Pneumonia, (3) Typhoidal state

Diagnosis.—In epidemics, diagnosis often simple Diagnostic methods are –

Suspected flesh – Lease on slide and examine by hand lens or microscope for larval forms

Parasites in human fæces—Dilute fæces in conical glass: examine sediment against black background for minute parasites; under microscope identify by 'cell body'

Excise small slips of biceps or deltoid of patient and examine.

Eosinophilia

X rays show calcified cysts

DIFFERENTIAL DIAGNOSIS

TYPHOID –In trichiniasis no headache, no splenic enlargement no spots, but pain and swelling of muscles and œdema. Also eosinophilia

RHEUMATIC FEVER – Distinguish by gastro intestinal stage.

BERI BERI may simulate closely No eosinophilia.

Treatment.—Indication is to empty intestine early in order to discharge worms Give calomel in large doses, e.g., gr ij. t d s. Glycerin advocated in large doses to dehydrate worm, doubtful value. Anthelmintics are useless No drugs affect the muscle trichinellæ. Muscle pain needs morphia

Prophylaxis.—Thorough cooking of all pork is best, and is efficient prophylactic measure, with rare exceptions.

In herds of hogs, measures advised are (1) Destruction of rats, (2) Uncooked offal of hogs not to be used as food (3) Examination of flesh in the abattoirs

3. ANKYLOSTOMIASIS.

(*Hookworm Disease. Uncinariasis*)

Synonyms.—In Europe, *Ankylostomiasis*. In America, *Hookworm disease* or *Uncinariasis*. Popular terms: miners' anæmia, tropical chlorosis, tunnel disease (from St Gothard Tunnel outbreak)

Geographical Distribution.—In tropical and subtropical countries, widespread. In parts of India and Porto Rico 60 to 95 per cent of population affected. Very prevalent in Southern States. Small epidemic in Cornwall in 1900, due to miners returning from abroad: studied by Haldane and Boycott. Parasite needs warmth and moisture.

Parasite. Two principal sub-groups of *Uncinaria*: (1) *Ankylostoma duodenale*, in Old World; (2) *Necator americanus* or *Uncinaria americana* (hookworm) in New World. Both are small, cylindrical nematodes.

1. ANKYLOSTOMA DUODENALE. Mouth is large orifice with two pairs of hook-shaped ventral teeth. Male length 10 mm.; at posterior end is an expansion the caudal bursa. Female length 10 to 18 mm.
2. NECATOR AMERICANUS. Differs from first in having 4 sharp lancets external to mouth on dorsal aspect, also simple tooth and pair of semilunar plates in place of hook-shaped teeth. Other slight differences.

OVA.—Characterized by segmentation within capsule, usually 4 or 8 cells when examined from fresh fæces. Occur in enormous numbers. Size 60 to 75 μ by 35 μ.

LARVÆ. Embryos may be born one to two days after leaving body, depending on warmth and moisture. Embryos then moult twice, after which they are infective. Can occur within four to five days. May live for months subsequently. Development most favourable in fæces mixed with earth.

Mode of Infection.—Domestic animals are not infected. Infection of human being occurs:—

1. THROUGH THE SKIN (Looss, 1898). Usual method. Larva penetrates skin, enters veins, passes through heart to lungs, escapes into bronchi, passes up trachea and down œsophagus to stomach and intestines, course occupying seven to ten days. (*See* ASCARIS LUMBRICOIDES, p. 191.) In the intestine larva moults again, and then matures. Ova are present in fæces in about seven weeks from entry.

   'GROUND ITCH' occurs at site of entry of larvæ. Vesicular eruption becoming pustular. Commonly under toes. In miners often on arms and hands. Heals in one to two weeks.

2. BY THE MOUTH. Rare. By water supply, infection of fingers.

MODE OF ACTION IN HUMAN BODY. The adult worm lives in the jejunum: by its teeth and lancets it pierces the mucosa and sucks blood. Probably also secretes hæmolytic toxin from head glands and diminishes coagulability of blood.

Morbid Anatomy. In mucosa of jejunum, ecchymoses and erosions present worm often attached in centre Also oozing points, probably vacated by worms In long standing cases, mucosa pigmented and infiltrated Often blood cysts, length ½ to 1 inch, containing one or two worms Intestinal contents often blood stained (though not fæces) Fatty degeneration of heart and other organs, if anæmia advanced Body is not wasted

Symptoms. Very variable In infected districts, subjects may be grouped into (1) Carriers' no obvious symptoms (2) A large group physically and mentally 'below par', without actual illness, but symptoms is below in varying degree May include a large percentage of the population especially children. (3) Those with serious symptoms, only a small percentage of infected

ESSENTIAL SYMPTOMS (i) *Anæmia*, with *œdema* and *lethargy* *Eosinophilia* (see below) (ii) *Digestive troubles* *Epigastric pain and tenderness* very constant, even in mild cases In severe forms anorexia and, characteristically *perversion of appetite*, especially dirt eating (pica' geophagy') Wasting not common, usually plump Bowels variable (iii) *Mental inertia* Listless expression Lack of concentration (iv) *In children* Under development, small stature, puberty delay of growth may continue until twenty five years

No enlargement of glands, spleen, or liver

OTHER SYMPTOMS *Fever* Variable, often transient rises *Blood changes* (a) Erythrocytes 'Secondary anæmia'. Hæmoglobin to 50 per cent Colour index low. Changes in form of red cells slight (b) Leucocytes *Eosinophilia* 15 to 25 per cent in recent infections often higher No leucocytosis *Parasites* Not in blood but present on testing 'Ground itch' Occur during infection

Duration. Chronic often many years Rarely acute

Termination in Severe Infections.– Anæmia extreme with usual sequelæ Œdema Serous effusions, Death from exhaustion or intercurrent disease

Diagnosis. –In infected districts suggested by anæmia, especially with history of 'ground itch' and in children associated with under development and physical and mental inertia Examine (1) *Fæces for ova* (following ingestion of thymol) often segmented, 4 or 8 cells, or more complete embryo (2) *Blood* for eosinophilia

Treatment. Essential treatment is to evacuate parasites from intestine When this is accomplished, recovery is good Test of success is absence of ova from stools during three weeks The following drugs have been extensively and successfully used —

OIL OF CHENOPODIUM – In meshly-filled hard gelatin capsules (1 c.c.) repeated one hour later Aperients two hours after. Repeat in ten days Cheapest and efficient.

THYMOL Method Saline purgative at night Following day. thymol at 6 a.m., repeat at 8 a.m., saline purgative at 10 a.m.

Ankylostomiasis—Treatment, *continued.*

Repeat weekly until cured. Dose of thymol: under five years, 7 gr., increasing to 30 gr. for active adult.

Precautions.—No alcohol or oil to be given during treatment: thymol is freely soluble in these, but only soluble 1 in 500 in water. Administration of such results in absorption o thymol, with vertigo, delirium, and occasionally fatal syncope. Contra-indicated in advanced cases, or with nephritis, or cardiac weakness.

EUCALYPTUS OIL.—Prescription (for adult):—

℞ Ol. Eucalypti	♏xxx	Ol. Ricini	ʒx
Chloroformi	♏xl		(Philips)

Divide in two portions and give as with thymol. Is safer than thymol. Repeat several days.

BETA-NAPHTHOL.—Three doses of 15 gr. at intervals of one hour, followed by purgative.

ANÆMIA requires usual treatment.

Prophylaxis.—Important measures are: (1) Disposal of fæces. Special care in mines. Population educated to use privies. (2) Pure water supply In absence of this, water to be boiled. (3) Children to wear shoes and stockings.

The Rockefeller Institute has instituted an anti-hookworm campaign in infected districts in America with marked success.

4. FILARIASIS.

Infection by *Filaria bancrofti* may result in obstruction of lymphatic vessels, of which the chief symptoms are chyluria and elephantiasis.

Geographical Distribution.—Widespread in tropics and sub-tropics. Occurs in Southern States. In Samoa affects nearly half the population.

Parasite.—

ADULT PARASITE.—Hairlike worm, length 2 to 4 inches, in many coils. *Site in body*: In thoracic duct, lymphatics, or glands, often in varices: never seen during life. Life: possibly years.

EMBRYO.—About 0·3 mm. × 0·01 mm. Structure slight. Is contained in a 'sheath', which *it does not fill* at the ends, and in which it moves back and forwards. Present in peripheral blood.

Life Cycle.—

1. *Mosquitoes* are intermediate host. Withdraw embryos from definitive host when feeding. In stomach of mosquito, embryo ruptures sheath, reaches thoracic muscles, and there undergoes development for twelve to twenty days Hence it passes to base of proboscis. When mosquito feeds, larva bursts from base of proboscis (not through salivary glands), escapes on to skin, and penetrates it near, but not at, puncture.

2. *In man*, these larvæ reach lymphatics, mature and produce embryos, which pass through lymphatics to veins and into peripheral circulation

Periodicity. Embryos are present in blood only at night, about 6 p.m. to 8 a.m. Appearance in blood precedes sleeping hours, but if man sleeps in daytime embryos are said to appear then. Connected with nocturnal habit of usual intermediate host, viz., the mosquito *Culex fatigans*

NON-PERIODIC FILARIA. In Fiji and islands near, embryos are present also during day. Parasite is identical, but intermediate host is the mosquito *Stegomyia pseudscutellaris*, which feeds by day

Pathology. Embryos are harmless; being breadth of red cells, can pass through capillaries without blockage; often present in man without symptoms, and in animals. Infection has no direct effect on blood-cells. Symptoms follow blockage of lymphatics by adult worms, or possibly by ova prematurely expelled. Results are:

1. Lymphatics enormously dilated round kidney, bladder, etc., forming varicose masses, and containing chyle. Thoracic duct may be stenosed. Groin glands often enlarged. Rupture of varices into urinary system causes *chyluria*. Parasite may be dead previously and no embryos in blood, but the latter are often present.
2. *Solid edema of elephantiasis*. Blockage of lymph vessels alone does not produce this; confirmed experimentally by ligature of animals; inflammation always occurs. This occurs in recurring elephantoid fever with lymphangitis, probably of septic origin.

RELATIONSHIP OF ELEPHANTIASIS TO FILARIASIS. Embryos are not present in blood, or only to less extent than in unselected persons. Relationship is inferred from: (1) Geographical distribution identical; (2) Both are lymphatic diseases with recurrent lymphangitis; (3) Elephantiasis is common sequel of, or coexists with, lymphangitis caused by certainly filarias.

Symptoms.

1. CHYLURIA. Passage of milky urine, usually blood-stained. No symptoms, or may be pain in back and pelvis. Occurs intermittently, at intervals of weeks or months, over many years, but if frequent, anæmia may result. *Urine* clots on standing. The pink coagulum contracts and expresses milky fluid. Later layer of fat globules may form on surface. Clots with ether. Embryos often present in blood and urine.
2. ELEPHANTIASIS.—*Legs* most commonly affected, especially below knee. Scrotum not infrequent. Enlargements often enormous. Mammæ and arms less frequent. Slow and painless. Fluid is lymph, not chyle.
   Elephantoid fever. Recurrent attacks of fever, pain and swelling in limb and lymphangitis. After attack, limb remains larger. Embryos not present in blood.

Filariasis—Symptoms, *continued.*

OTHER CONDITIONS are: '*Lymph scrotum*': Lymphatics dilated and varicose over scrotum: chyle discharges if vessels rupture Subsequent elephantiasis common. '*Varicose groin glands*': Chronic, bilateral: frequently with lymph scrotum. *Lymphangitis.*

Diagnosis.– Generally simple, either by symptoms or by presence of embryos in blood With chronic enlarged glands in groin in patients from tropics and subtropics, examine blood.

Treatment.– No drug kills the adult worm or affects the embryos.

CHYLURIA – Rest, purge, dry diet, avoid fats Disappearance of chyle does not prove rupture is healed: can be tested by drinking glass of milk and watching for reappearance of fat

ELEPHANTIASIS – Carefully protect from injury and sepsis

ELEPHANTOID FEVER – Rest, purge cooling lotion to sites Bandage firmly subsequently

ELEPHANTIASIS OF SCROTUM. May be removed by operation

'GROIN GLANDS' and 'LYMPH SCROTUM' Operation inadvisable. In latter, elephantiasis of leg may follow.

General Characteristics. (1) Embryos only are found in blood, (2) Sheath of embryo is incompletely filled, (3) Nocturnal periodicity, (4) Symptoms mainly (*a*) chyluria, embryos often present, (*b*) elephantiasis, embryos rarely present

Varieties of Filaria.

Of *Filaria sanguinis hominis* (embryos present in blood, three species are known.

F. bancrofti Described above Sole cause of symptoms of chyluria and elephantiasis.

F. diurna: Present by day Possibly larval form of *F. loa* (habitat commonly beneath conjunctiva) West Africa

F. (*Acanthonemonema*) *perstans* Embryos alone known No periodicity

Other species have also been described Guinea worm is a filaria

5. DRACONTIASIS.

(*Guinea worm Disease*)

Infection by *Dracunculus* (*Filaria*) *medinensis.*

Geographical Distribution. Certain parts of India and Africa, especially West Africa Districts are fairly limited, probably by distribution of intermediate host

Parasite.—*Female guinea worm* is about 80 cm long by 1·5 mm. in breadth shape, cylindrical On tail is a minute hook Uterus occupies almost entire body, is packed with embryos, which are discharged by prolapse of the uterus through the mouth Of the male, little is known: life probably much shorter and dies after coitus Parasite enters human body by mouth in drinking water and reaches stomach Female penetrates intestine after

impregnation reaches subcutaneous connective tissue develops, and then wanders down in tissues usually to foot or ankle where it lies subcutaneously Here the skin is penetrated a small vesicle forming and bursting through this erosion the head can protrude, and embryos are discharged when site is in contact with water After all embryos are discharged worm usually leaves host Occasionally worm becomes calcified under skin

Intermediate host in water is a minute *Cyclops* a crustacean Embryo after entry undergoes certain changes and is then infective Many features are related to this host The guinea-worm travels to parts where it can discharge its embryos into water, in native water carriers it often appears on back where bag rests douching site of head produces a discharge of fluid containing numerous embryos The life of the female guinea worm is about a year probably corresponding to some development of the *Cyclops*

Treatment.—

1 After head appears or when worm is within reach subcutaneously inject its body with perchloride of mercury 1 1000 worm dies in twenty four hours and can be withdrawn

2 Douche with warm water as worm protrudes roll it on a small stick Repeat daily Danger is that worm may break when embryos are discharged into tissues and *extensive tissue suppuration follows* No traction must be used as worm resists, probably by hook on tail

By douching discharge of embryos is complete in fifteen to twenty days and worm is then dead and usually leaves host spontaneously

6. TRICHOCEPHALUS DISPAR.

(Whip worm)

Synonym Trichuris trichiura Inhabits caecum and large intestine of man Distribution probably universal and occurrence frequent

WORM Length about 2 inches Shape resembles a whip anterior portion very thin and posterior portion thick, being in female straight and in male coiled

OVA Oval dark brown Characteristic *lighted area* or *bulging knob* at each end

MODE OF INFECTION Direct by water No intermediate host

SYMPTOMS None by which infection can be recognized Possibly causes anæmia, but little known

Section I.—Specific Infectious Diseases, *continued.*

E. INFECTIOUS DISEASES OF UNKNOWN OR DOUBTFUL ETIOLOGY.

CHAPTER XXVI.

SMALL-POX.

(*Variola*)

An acute infectious disease characterized by an eruption which passes through successive stages of papule, vesicle, pustule, and crust, with subsequent scarring.

Prehistoric in Old World: introduced into America in 16th century.

Etiology.—*Susceptibility* almost but not quite universal. Extremely contagious: almost invariably contracted by unvaccinated persons on first exposure to infection. *One attack* does not always protect for life, but second attacks are very rare.

AGE.—All ages equally susceptible, but mortality very high in young children.

SEX.—Equal in males and females.

RACE.—Negroes especially susceptible.

CLIMATE AND SEASON. Of little influence; more prevalent in tropics. In temperate regions, more frequent in winter than summer.

Epidemics vary greatly in severity: some recent ones very mild.

Morbid Anatomy. *Pustules* present on skin, tongue, and palate, often on larynx: may extend to stomach: none on trachea, but may be ulceration.

Spleen enlarged. Lymphatic glands become enlarged.

In hæmorrhagic forms, hæmorrhages occur in all tissues and organs.

FORMATION OF A POCK. Degeneration of cells commences among the prickle-cells. The cells liquefy, and lymph is also exuded, a vesicle resulting. The *vesicle is multilocular*, owing to persistence of trabeculæ, and does not collapse from a single prick. *Umbilication* is due to changes being more advanced at periphery. The *pustule is unilocular*, trabeculæ being destroyed.

SPECIFIC VIRUS. Unknown.

Mode of Infection.—The virus enters by mucous membrane of nose, mouth, or respiratory tract. Communicated by: (i) Infected persons; (ii) Infected articles and fomites; (iii) Third persons; (iv) Inoculation, now illegal (*see* VACCINIA, p. 209).

OCCURRENCE OF INFECTIVITY.—Infected persons undoubtedly infectious from commencement of rash *until skin entirely clear*. Greatest during pustulation, but also infective in *pre-eruptive period and during prodromal rashes* (not fully proved). *Dried scales* are main source of infection, virus spreading aerially and infecting articles and persons. Pustules under skin of palms, soles, or nails may not rupture, and must be cut away or infectivity remains. Dead bodies are infectious. *Virulent types* may follow infection from the mildest varioloid. Entering a room is sufficient for infection.

AERIAL TRANSMISSION over considerable distances is possible, but not fully proved. Cases occur near isolation hospitals, but some are possibly direct infection.

DURATION OF INFECTIVITY.—Until scabbing has completely ceased and all crusts separated.

QUARANTINE PERIOD FOR CONTACTS.—Sixteen days. (Period usually fixed at sixteen days, but a few undoubted cases are recorded with incubation period of twenty days.)

INCUBATION PERIOD.—Nine to fifteen days, usually twelve (fairly constant). Extreme limits possibly five to twenty-one days, or longer. No symptoms.

Varieties of Small-pox.

(1) VARIOLA VERA.—(i) Discrete; (ii) Confluent.

(2) HÆMORRHAGIC SMALL-POX.—(i) Black small-pox, purpura variolosa; (ii) Hæmorrhagic pustular small-pox.

(3) VARIOLOID.—Small-pox modified by vaccination.

VARIOLA VERA.

Clinical Stages.—(1) Invasion; (2) Initial rashes; (3) True eruption; (4) Desiccation.

Stage of Invasion.—*Prodromal period*: onset usually sudden; in adults rigors or chills; in children convulsions. *Characteristic* initial symptoms are: (i) *Frontal headache*, absence rare; (ii) *Vomiting* and epigastric pain; (iii) *Pain in back*; often also elsewhere. All three often intense.

Temperature on 1st day, 103°. *Pulse* rapid. *Constipation*. Tongue furred. Breath offensive. Throat often sore. *Restlessness*, insomnia, and often delirium. Prostration may be severe. Skin usually dry, but may be sweats. Respirations may be rapid.

Severe initial symptoms may be followed by mild attack: mild initial symptoms never by a severe attack.

Initial Rashes.—Usually on second day. Frequency varies greatly in different epidemics; up to 15 per cent of cases.

TYPE OF RASH.—(1) Scarlatiniform. (2) Morbilliform. These may be general, or roughly of 'bathing-drawers area'. (3) Petechial, especially 'bathing-drawers area'. Rarer types: urticaria, purpura. Petechial and generalized rashes are usually followed by severe or hæmorrhagic symptoms.

Small-pox—Variola Vera, *continued.*

Duration of initial rashes generally two days, occasionally five days: usually fade entirely just before true eruption, but may overlap.

Stage of Eruption.—This stage is here described under two divisions, *discrete* and *confluent.*

Discrete Form.

In this form the pocks remain separate from each other.

ONSET OF ERUPTION.—Fourth day. Appears first on forehead, back of wrists and hands. Often at same time in mouth and fauces. Rash spreads on face, trunk, and extremities: last on lower extremities, soles, and palms: development occupies about three days.

CHARACTER OF ERUPTION.—Successively macule, papule, vesicle, pustule, and crust. Early stage of spots: *bright red macules*, diameter $\frac{1}{16}$ inch, disappear on pressure; in a few hours become *papular*, 'like shot in the skin'. On fifth to sixth day of illness *vesicles* form, with clear summits, *umbilicated*, diameter $\frac{1}{8}$ inch, opaque. On eighth day become *pustules*: spots swell, become round-dome-shaped, *umbilification lost.* Injected areola surrounds pustule. Skin much swollen. This *maturation* commences on face and spreads.

DISTRIBUTION OF ERUPTION.—Spots most numerous on face, scalp, lower extremities, and upper back; least on abdomen, chest, and back; may be many thousand spots. Face, mouth, larynx, and pharynx very painful.

SYMPTOMS.—With onset of rash, temperature and symptoms subside. With maturation at 8th day, general symptoms return, and *'secondary fever'* occurs. *Itching* extreme, and great pain from swollen skin. *Face* especially painful. *Eyelids* swollen and closed. Mouth dry and deglutition painful. Thirst extreme. *Delirium* slight or absent, but in severer cases may be acute and suicidal. Odour often distinctive, but more marked later.

STAGE OF DESICCATION. *About tenth day* pustules commence to rupture and pus exudes. Subsequently they dry rapidly, first on face. Temperature falls gradually and convalescence begins. *By fourteenth* day decrustation advanced on face. Scabbing continues during third and fourth weeks.

TEMPERATURE.—(1) High on first day, 103° to 104°; (2) Falls with true rash; (3) Rises again with maturation, 'secondary fever'; (4) Commences to fall between tenth and fourteenth days.

LIVER AND SPLEEN.—Not palpable. Bowels costive.

EITTING.—Slight in this form.

UNFAVOURABLE CASES.—After the eighth day typhoid state develops with extreme prostration: heart fails. Death usually twelfth to fourteenth day.

Confluent Form.

Pocks coalesce. Initial symptoms usually more severe.

ONSET OF ERUPTION.—Fourth day or earlier. The earlier the eruption the more often is it confluent.

CHARACTER OF ERUPTION —Passes through same stages as discrete form In the milder cases papules are early discrete, and confluent only when pustular In more severe cases pustules are very close, skin greatly swollen and hyperæmic With onset of rash, temperature and symptoms subside, but not so completely as in discrete form.

On eighth day pustules form, and coalesce, large superficial abscesses result Pustules in mouth, larynx, and pharynx Cervical glands much swollen Fœtor extreme. *General symptoms return* in marked degree, and condition is pitiful *Temperature* high, pulse rapid, thirst marked, delirium frequent.

STAGE OF DESICCATION —Pustules break and exude pus, or may desiccate unruptured Scabbing occurs in third and fourth weeks the crusts are very adherent and may require treatment Pocks which remain unruptured under skin of palms, soles, and nails must be cut away

DISTRIBUTION OF ERUPTION —*Confluence* extreme on face, feet, and hands On limbs scattered patches On trunk, spots always discrete The eyes are closed skin markedly swollen The [illegible] life varies with number of spots on face

UNFAVOURABLE CASES Death results from (1) Delirium, prostration, and cardiac failure tenth to twelfth day, (2) Hemorrhages *see below*, HEMORRHAGIC SMALL-POX, (3) Pneumonia during convalescence

FAVOURABLE CASES Improvement commences about twelfth day Desiccation occurs and symptoms subside

HÆMORRHAGIC SMALL-POX.

Occurs in two forms (1) *Black small-pox or purpura variolosa*, (2) *Hæmorrhagic pustular small-pox*

Purpura Variolosa.

FREQUENCY Varies in different epidemics Most common in healthy adult males Rare in children and vaccinated persons

INITIAL SYMPTOMS As in other forms, but always severe

ERUPTION —Appears on second, third, or fourth day petechial from onset, with diffuse hyperæmia Often commences in groins, spreads rapidly with extensive subcutaneous and cutaneous hæmorrhages, and becomes universal Usually hæmorrhages from mucous membranes, hematuria, hæmatemesis, hæmoptysis etc

CONDITION Becomes appalling face swollen, conjunctival ecchymoses, entire skin of purple hue, bloody saliva and foul breath, extreme prostration and collapse Mind may be clear to the end

DEATH on third to fifth day, rarely sixth recovery never occurs Two groups may be distinguished, (a) Prodromal rash, usually petechial, followed by the purpuric eruption, (b) Eruption purpuric from onset The characteristic pustular eruption is not present, and in a sporadic case the diagnosis is very difficult.

Small-pox—Hæmorrhagic Small-pox, *continued.*

Hæmorrhagic Pustular Small-pox.

Commences as severe variola vera. Hæmorrhages commence in vesicular or pustular stage; the earlier the onset the more severe the condition. Blood appears first in areolæ surrounding spots, and spreads rapidly. Hæmorrhages from mucous membranes common.

DEATH on seventh to ninth day; recovery occasionally occurs (In discrete form, hæmorrhages into spots on legs may occur if patient gets up too early.)

Blood Changes. Marked polynuclear leucocytosis in all forms.

VARIOLOID.

Modified form occurring in vaccinated persons. *Onset* abrupt. *Initial symptoms* may be severe as in other forms. *Rash*, as papules, appears on third or fourth day. With eruption, temperature and symptoms subside. No secondary fever occurs. Stages of vesicle and pustule are short. Pitting rare. Within five years of vaccination variloid is rarely severe, but occasionally is fatal.

NOTE.—These cases are infectious, and virulent types may occur in the infected.

MILD AND ABORTIVE FORMS.

Some recent epidemics have been very mild; the initial symptoms may be severe, but the number of spots is usually small and the constitutional disturbance slight.

Possibly several types of virus exist. 'Amaas strain' is connected by epidemiologists with outbreaks in America, Australia, and other parts, in which mortality is 0·5 to 5 per cent. 'Alastrim' (Jamaica) has resemblances both to variola and varicella, and also differences; relation to 'Amaas' also disputed; mortality very low.

VARIOLA SINE ERUPTIONE. Occasionally recognized in an epidemic. Initial symptoms only.

WART POX. Vesicles abort at fifth or sixth day.

COMPLICATIONS.

BRONCHOPNEUMONIA present in all fatal cases.
DELIRIUM AND COMA. Convulsions common in children.
LARYNGITIS may be dangerous from œdema of glottis, aspiration pneumonia, or necrosis of cartilages.
ALBUMINURIA frequent, but nephritis rare.
CONJUNCTIVITIS common, but usually avoidable with care.
KERATITIS not uncommon in confluent form.
SEPTICÆMIA may develop in pustular stage or later.

Sequelæ.—

PITTING.—Especially on face in confluent form.
BOILS AND ABSCESSES.—Very frequent and troublesome. Cellulitis and erysipelas occasionally during scabbing.

Rare: Post-febrile insanity. Arthritis. Pseudotabes (*ataxie variolique*), very rare.

A secondary eruption occasionally occurs during desquamation, so-called 'recurrent small-pox'.

PROGNOSIS.

Depends on: -

1. VACCINATION —In vaccinated persons mortality very low. A few per cent in most unfavourable circumstances. Depends also on number of vaccination marks. With successful re-vaccination, mortality nil (*See* VACCINIA)
2. AGE In unvaccinated persons mortality highest in infancy, diminishes in childhood, and then increases progressively Average about 25 to 35 per cent
3. CLINICAL TYPE Hæmorrhagic form practically always fatal Confluent form, mortality about 50 per cent Discrete form about 5 per cent. Intermediate types between discrete and confluent occur, with varying mortality
4. SPECIAL SYMPTOMS - Prognosis depends especially on *amount of eruption on face* Unfavourable symptoms also are delirium, high temperature, laryngitis, and pulmonary affection especially in children
5. VIRULENCE OF EPIDEMICS This varies very greatly (*See* p 206)

DIAGNOSIS.

During epidemics initial symptoms are usually diagnostic In sporadic cases diagnosis usually impossible before eruption

Confusion arise with -

1. INITIAL RASHES May simulate scarlet fever or measles, Note in *small pox* distribution of rash and initial symptoms
   - In *scarlet fever*, sore throat rash more extensive and persistent
   - In *measles*, coryza and conjunctivitis, long prodromal period. Koplik spots
2. VARICELLA. Main points of diagnosis are -

   IN VARICELLA Rash (i) Usually on first day; (ii) Appears first on chest or back (iii) Abundant on trunk and scalp, (iv) Not shotty at onset, and no umbilication; (v) Coelæ slight, (vi) Successive crops on several days; (vii) Spots present simultaneously in all stages of evolution; (viii) Temperature and malaise slight

   IN SMALL-POX (i) Longer period of invasion, (ii) Initial rashes, (iii) True eruption first on forehead, wrists, and hands, (iv) Shotty feel, umbilication, (v) Temperature and symptoms severe

   Mucous membranes affected in both. In epidemics a severe case often decides diagnosis
3. HÆMORRHAGIC SMALL-POX -Diagnosis from hæmorrhagic scarlet fever or measles: often impossible if sporadic, but rash more marked on mucous membrane in small pox
4. CEREBROSPINAL FEVER. Cerebrospinal fluid is diagnostic

LESS COMMONLY. —*Influenza* no rash; initial symptoms similar

Rubella: rash on first or second day: symptoms very slight.

Small-pox—Diagnosis, *continued.*

Typhus no rash on face; no fall of temperature with onset of rash. *Erythemata, e.g.*, from food and fish: onset extremely rapid; diffuse. *Pustular glanders* nasal discharge, character of eruption, symptoms out of proportion to rash.

PROPHYLAXIS.

VACCINATION and re-vaccination repeated after exposure to infection (*See* VACCINIA, p. 209.)

DURING AN EPIDEMIC.—General vaccination. Complete isolation of contacts. Great care in diagnosis of mild cases. (*See also* MODE OF INFECTION.)

TREATMENT.

Isolation in special hospitals imperative. No specific treatment exists. Varioloid and discrete cases require little special treatment. In severe cases treatment especially for eruption and constitutional symptoms.

GENERAL HYGIENE. Bed, fresh air, plenty of fluid, milk diet. Water bed if necessary. *Nose and mouth* swabbed gently or syringed (*see* SCARLET FEVER). Ice to suck when mouth affected.

INITIAL SYMPTOMS.—*Pains* need opium. *Vomiting* ice and champagne, and opium. *High temperature* hydrotherapy, especially cold packs.

ERUPTION.—Cut hair short. In early stages lint mask over face. Moisten with cold water or 2 per cent carbolic. Cover with oiled silk. Itching in all parts is relieved by cold and moisture. When crusts form, skin must not be allowed to dry. Best for face is a mask of thin linseed poultice covered with a little vaseline and frequently renewed. For body moisten with glycerin or vaseline. For unruptured pustules, especially under nails, incise and treat aseptically.

All treatment with oil, liniments, etc., useless, and probably delays separation of crusts.

BATHS.—Continuous warm bath most valuable. Should be used in all cases of suppuration, confluent spots, or toxæmia, and also to hasten separation of crusts.

EYES.—Treatment of great importance. Bathe with boracic lotion, smear edges of eyelids with vaseline. Usual treatment for keratitis.

HÆMORRHAGIC CASES.—No treatment of any effect. Hæmostatics useless.

VARIOUS SYMPTOMS AND COMPLICATIONS. *Delirium and sleeplessness* need opium. For *cardiac weakness* give alcohol and stimulants. Great swelling of tongue may need incisions. Laryngitis tracheotomy may be necessary.

PROTECTION FROM LIGHT.—Maturation is less when spots guarded from light. Finsen's red light has been tried. Result doubtful.

CONVALESCENCE. Give frequent baths to hasten separation of crusts. Convalescence usually rapid. *Boils*, open on formation, and give continuous warm bath.

KOPLIK'S SPOTS *Minute white specks* surrounded by red areolæ on buccal mucous membrane, most commonly at level of lower second molar or milk molar. Numbers very variable, and distribution may be extensive. Areolæ frequently absent, as noted by Goodall. Appear usually on second day. Disappear rapidly after eruption comes out. Presence very constant and pathognomonic (difficult to see by artificial light).

STAGE OF ERUPTION. Symptoms increase until fourth day, when the eruption appears.

ORDER OF ONSET OF ERUPTION. Earliest on temples, on forehead at margin of hair, and behind ear. Spreads rapidly in a few hours over face, trunk, and finally limbs. Feet and hands are last affected. Maximum in one to three days. Amount of eruption varies, but some normal skin is always present.

CHARACTER OF RASH. *Large stage*: small red spots like flea bites, or diffuse redness, disappearing on pressure. *Typical eruption* develops a few hours later: irregular, blotchy, crescentic patches of erythema, dusky red, edges red, raised to the finger, do not disappear entirely on pressure. The rash fades with cold, and becomes more marked with warmth.

PRODROMAL SYMPTOMS. Symptoms increased with eruption, but continue till fifth or sixth day. *Bronchitis* develops, scattered rhonchi and râles in lungs. *Epistaxis* common. Diarrhœa occasionally. *Fever* rises to maximum (104°) with appearance of rash. Pulse and respiration rapid. Dry cough. Restlessness and insomnia; may be delirium.

DURATION OF ERUPTIVE STAGE. Three or four days, rarely six. *Commences to fade* in twenty-four hours, in order of appearance; may fade on face, but persisting on limbs; last on hands, wrists, ankles, and knees. Desquamation of pinkish branny cells, varying with roughness of rash; duration about two days.

TEMPERATURE CURVE. Typical course: moderate pyrexia on first day (102°); fall on second day (100° to 101°); rises to maximum at onset of rash (104° to 105°); falls rapidly as rash commences to fade, and normal about seventh day from onset. Delayed by pulmonary or other complications.

VARIATIONS IN RASH. *Petechiæ* occur with hæmorrhagic tendency, usually near joints. The typical rash, varied when confluent, may resemble scarlet fever.

CONVALESCENCE. Rapid in absence of complications. Usually no symptoms in ten days from onset. Cough persists longest.

The Blood. No leucocytosis except with complications. Note.—Leucocytosis is said to be present during period of incubation.

Variations in Clinical Type. All are rare.

MILD FORMS. Catarrhal symptoms absent; convalescence by fifth day.

MORBILLI SINE MORBILLIS. Clinical symptoms without an eruption. Occurs in (1) Mild cases (rash may be transient).

Measles—Variations in Clinical Type, *continued*

(2) Severe cases usually cachectic patients typhoidal condition develops, with collapse and death absence of eruption may be due to death occurring before eruptive period or to rash fading through failure of circulation (the layman's 'rash driven inwards') Recognizable in epidemics by Koplik spots and by exposure to contagion and transmission to others

HÆMORRHAGIC MEASLES ('Black Measles') Extremely rare Occasionally in epidemics Widespread hæmorrhages of skin and mucous membranes, marked toxæmia Death second to sixth day Many epidemics of 'black measles' formerly described were probably erroneous diagnoses, e g small pox

Relapses.—Very rare

Complications. Severe complications are

1. BRONCHITIS AND BRONCHOPNEUMONIA *Bronchitis* is practically constant Usually first evident during eruption *Bronchopneumonia* serious and not uncommon is cause of most deaths convalescence slow Other respiratory complications *Laryngitis* mild form almost constant severe form followed rarely by œdema glottidis pseudomembranous laryngitis or pachondritis *Lobar pneumonia* rare
2. STOMATITIS AND NOMA Mucous membrane of mouth constantly affected in some degree May be severe ulceration serious *Cancrum oris* or *noma* is almost limited to measles always fatal may affect vulva
3. OTITIS MEDIA Not uncommon Mastoid abscess meningitis, etc may follow
4. DIARRHŒA Common during eruption
5. CONVULSIONS Serious when recurrent

Rare complications are Nephritis (transient albuminuria occasionally occurs during the eruption and is a definite hemiplegia with aphasia ... and usually permanent

Sequelæ. *Pulmonary tuberculosis* not uncommon high mortality *Chronic bronchitis* and recurrent bronchitis *Enlarged tonsils and adenoids*

Association with other Diseases. Common with other specific fevers, especially diphtheria (serious), scarlet fever, and whooping cough Owing to these conditions being common at same age as measles, exact relations of their association are still in dispute

Diagnosis. Difficulty may arise with —

SCARLET FEVER In measles (i) Longer prodromal period (ii) Affects mouth rather than throat (iii) Marked catarrhal symptoms and conjunctivitis, (iv) Rash blotchy, crescentic commences on forehead, and affects face, no circumoral pallor (v) No leucocytosis, (vi) Koplik spots diagnostic Desquamation branny only

RUBELLA.—(i) Shorter prodromal period (ii) Slight symptoms eyes clear, (iii) Occipital glands enlarged, (iv) Rash usually a punctate erythema.

URTICARIA.—This may suggest the rash of measles, but other symptoms absent.

SMALL POX.—Prodromal rash and early symptoms may resemble measles, and vice versâ.

Prognosis.—Immediate mortality practically confined to bronchopneumonia, normally invariably fatal, but rare diphtheria, high mortality, occasionally from diarrhœa, etc. Measles causes more deaths than the total of the other usual infectious fevers of childhood, and great care is necessary during convalescence. Mortality varies with age, being higher in infancy and in old age, with poor social conditions and environment, and also in different epidemics. Mortality in general about 3 per cent. Subsequent to attack, pulmonary tuberculosis may be fatal. Epidemics among unaccustomed populations have caused enormous mortalities. In the Fiji outbreak, deaths among adults were principally due to subsequent dysentery.

Prophylaxis.—Prevention of spread difficult, owing to long prodromal period and contagiousness in early stages.

Treatment.—Danger arises from the respiratory complications. Treatment aims at (1) Avoiding complications, (2) Treating symptoms, (3) Preventing spread of infection.

In ordinary cases no other treatment necessary.

GENERAL HYGIENE.—Bedroom temperature 63°, free ventilation, light covering, flannel on chest, bronchitis kettle in room containing tinct. benzoin co., aperient (calomel), no bath until rash subsides. For photophobia, screen from light.

MOUTH AND STOMATITIS.—Swab with diluted tincture of myrrh and borax.

COUGH.—If troublesome, treat as in acute bronchitis.

CONJUNCTIVÆ.—Bathe with boracic lotion.

GENERAL DISCOMFORT AND HEADACHE.—Antipyrin.

BRONCHOPNEUMONIA.—See ACUTE BRONCHITIS and BRONCHOPNEUMONIA. *Stimulants necessary.*

LARYNGITIS.—If severe, bronchitis kettle in tent. Fomentation over trachea (avoid blistering), usually besides with emetics. Tracheotomy rarely necessary, intubation only in hospitals. Remember diphtheria is possible; give antitoxin if doubtful, and swab throat for examination.

PYREXIA AND DELIRIUM.—Sponging, cold packs. Ice to head.

COLLAPSE, CYANOSIS with high fever.—Mustard bath.

DIARRHŒA.—Castor oil. Low diet. Bismuth and opium mixture. (*See* DIARRHŒA.)

VOMITING.—Peptonized milk. Bismuth.

SKIN AND RASH.—For itching: carbolized vaseline. During desquamation, rub with oil. Hot drinks or baths if eruption does not develop.

CONVALESCENCE.—In bed until temperature normal for a week. Out of doors after another one to two weeks. Special care of cough, if persisting, examine tonsils and adenoids; if necessary, send to dry high climate. Tonics and cod liver oil. Great care in the following winter.

CHAPTER XXVI

RUBELLA.

(German Measles Röthein Rose Measles)

A mild acute specific infection characterized by a rose pink papular or macular eruption appearing early, by enlarged glands in the neck, and slight constitutional disturbance A distinct disease neither measles nor scarlet fever protecting against it

Mode of Infection. Spread by direct contact Very infectious causing large epidemics Adults often attacked

DURATION OF INFECTIVITY Seven days after temperature normal Infectivity apparently commences two or three days before symptoms

QUARANTINE PERIOD FOR CONTACTS Twenty one days

Symptoms.

INCUBATION PERIOD Fourteen to twenty one days rarely up to twenty nine days

ONSET Slight malaise headache conjunctivitis and pyrexia

RASH Often the earliest symptom rarely later than second day

DISTRIBUTION Commences on face or front of trunk or limbs simultaneously Spreads to lower extremities in twenty four hours often fading from face Rarely on feet

CHARACTER Discrete numerous rose pink patches on trunk and limbs often confluent rapidly becoming indistinguishable here from scarlet fever Occasionally macules but smaller than measles

DURATION One to two days, rarely three disappears from feet last Leaves slight stain desquamation and rarely slight

OCCIPITAL GLANDS Enlarged Constantly palpable in cervical and mastoid Never suppurate

CONSTITUTIONAL SYMPTOMS Mild Loss of appetite headache and slight redness May be rash on soft palate Temperature often normal may rise to 104 for one to two days

Complications. Rare Secondary infection Mortality negligible

Diagnosis. From

SCARLET FEVER Rash may simulate scarlet fever on second day when faded from face and confluent on trunk but remains discrete on feet, constitutional symptoms slight marked pallor, no peeling

MEASLES -No coryza, prodromal symptoms, enanthema, conjunctivitis slight, rash is brighter tint

Treatment.— No special treatment necessary.

'Fourth Disease'.- Clement Dukes describes a condition lacking certain symptoms of, or in some degree varying from, rubella, measles, and scarlet fever Not generally confirmed

CHAPTER XXXII.

MUMPS.

(*Epidemic Parotitis*)

An acute specific infection characterized by swelling of the salivary glands especially the parotids.

Etiology. Widespread. Endemic in most towns. Large epidemics common.

- AGE. Mainly five to fifteen years; infants rare; adults not immune. Boys specially liable.
- SEASON. Prevalent in winter and spring.

Morbid Anatomy. Changes mainly inflammatory in connective tissue of glands, and but slightly affecting parenchyma.

Mode of Infection. By direct contact; exposure often very short.

- One attack protects.
- VIRUS. Not known. From the occurrence of orchitis, pancreatitis, etc., it appears to attack secretion glands with special predilection for the parotids.
- DURATION OF INFECTIVITY. Isolate for three weeks from onset; for enlargement of glands. Must be one week after swelling subsides.
- QUARANTINE. PERIOD FOR CONTACTS. Five weeks.

Symptoms.

- INCUBATION PERIOD. Usually ten to twenty-one days; rarely twenty-five.
- PRODROMAL. Malaise for one to two days; often absent.
- PAROTID GLAND. Swelling and tenderness commences *behind the angle of the jaw*, obliterating the lobe of the ear; *spreads forward over the face and downwards beneath the sternocleidomastoid*, doughy; skin may be red; pain on opening mouth; vomiting with degree of swelling and tension. When severe, *oedema* of neck and enlarged cervical glands. *Unilateral at first*; other side usually follows in one to five days.
- SUBMAXILLARY GLANDS. Usually enlarged; occasionally without parotid enlargement. *Lingual glands* less often.
- TEMPERATURE. About 101°.
- DURATION. Glands attain maximum in three to four days; subside in seven to ten days. Relapses rare.

Complications. *Rare*, except orchitis, but sometimes severe.

1. ORCHITIS.—In 30 to 40 per cent, especially young adults. Onset about eighth day, with fever and malaise; swelling of one or both testes, occasionally urethral discharge. Duration three to five days. *Atrophy may follow.* In epidemics, cases of orchitis occasionally occur without parotitis. *Ovaritis* may occur; suggested by pain and tenderness in lower abdomen and pyrexia.

Mumps Complications, *continued*

2 'CEREBRAL MUMPS' Delirium, great pyrexia may be coma and symptoms of meningitis Rare, but considerable mortality
3 ACUTE PANCREATITIS Pyrexia, epigastric pain and abdominal discomfort Rarely serious
4 PAROTID GLANDS Chronic hypertrophy (Possibly connected with carious teeth or oral sepsis)
5 DEAFNESS Rarely permanent Otitis media rare
6 SUPPURATION OF GLANDS Extremely rare
7 MASTITIS occasionally occurs

Various Rare Sequelæ.—Peripheral neuritis, paralyses affection of special senses nephritis

Diagnosis. Simple A septic parotitis may occur in conditions when mouth becomes dry (see INFLAMMATION OF THE SALIVARY GLANDS p 362) In glandular fever the salivary glands are not affected

Prognosis.—Mortality practically confined to 'cerebral mumps'

Treatment.—*Rest in bed* for ten days at least Purgative Mouth washes *Diet* jellies custards and semi solids swallowed more easily than fluids *Local treatment* if very tender hot or cold compresses as desired sprinkled with tinct opii or paint with glycerin and belladonna, cover with cotton wool Leeches relieve great tension *Orchitis* rest in bed wrap in cotton wool and support testes *Cerebral symptoms* ice cap to head

CHAPTER XXXIII

TYPHUS FEVER.

(*Gaol Fever Spotted Fever* and many local terms)

An acute highly contagious disease due to unknown virus conveyed by lice, characterized by sudden onset marked nervous symptoms and toxæmia rash and pyrexia terminating by crisis about fourteenth day Typhus and typhoid fever only distinguished in the 19th century

Etiology. Epidemics of enormous extent occur Endemic in Russia and the Balkan States Ireland has suffered heavily Also in Mexico and Eastern States of America Principally in temperate regions War famine poverty and dirt favour outbreaks In peace modern sanitary science can now control it Spreads more rapidly than any of the other great epidemic diseases . mortality among attendants is high

Morbid Anatomy.—No characteristic changes Ordinary changes of acute fever present • Rash is visible after death *Spleen* moderately enlarged

Mode of Infection.* Little understood until modern researches. NICOLLE 1909, discovered three essential facts:—

(1) *Blood infective* in febrile stages, also shortly before and after. Reproduces the disease in monkeys, and can be conveyed similarly to other monkeys.

(2) *Transmitted by lice.* Lice most infective five to seven days after feeding, and hence probably some development occurs. Infection is transmitted through eggs, and second generation of lice can convey infection.

(3) During epidemics, children may have rise of temperature with no other symptoms, and their blood be infective to monkeys. The disease may be kept alive by such cases, and this may account for epidemics apparently arising de novo.

RICKETTS and WIDER, on Mexican typhus, and ANDERSON and GOLDBERGER, on Brill's disease in the States, confirmed these results. The latter proved Brill's disease to be mild typhus.

Guinea-pigs can be infected, but apparently not other animals.

Recent epidemics in Serbia and other parts have been controlled by measures directed against lice.

Never [illegible] or [illegible] borne.

NATURE OF VIRUS.—Doubtful if a filter-passer. Ricketts found minute bodies in blood of typhus patients, and in stomach of typhus-fed lice; regards these as causal protozoa *Rickettsia prowazeki*.

SERUM. In convalescent patients is protective for a short time only. A protective serum has been prepared from guinea-pigs.

WEIL-FELIX REACTION.—A bacillus of the *Proteus* group (*proteus X 19*) has been isolated repeatedly from the excreta of typhus patients. While undoubtedly not the causal organism, this is agglutinated in a high dilution by the serum of typhus patients from about the sixth day. Priority for this reaction is due to W. J. Wilson (Belfast).

ISOLATION OF PATIENT. Four weeks.

QUARANTINE PERIOD. Fifteen days.

Symptoms.

INCUBATION PERIOD. Usually about twelve days, but very variable. Limits five to fourteen days; possibly three weeks. Occasionally slight malaise for a day or two.

CLINICAL STAGES. (1) Invasion, first to fifth day. (2) Nervous excitement and eruption, fifth to tenth day. (3) Nervous prostration, tenth to fourteenth day. (4) Crisis.

1. Stage of Invasion.—

ONSET.—Abrupt.

RIGORS. Common. Chills may recur in 24 hours.

PAINS. In back and legs, especially thighs.

HEADACHE AND NAUSEA.—Vomiting not uncommon.

MENTAL SYMPTOMS. Onset early, commence with sleeplessness. *Early prostration.*

* Muir and Ritchie *Bacteriology*, [illegible] edition.

Typhus Fever Symptoms, *continued*

EXPRESSION Dull, becoming vacant Face flushed with earthy hue (*facies typhosa*)

TEMPERATURE High at onset rises steadily to maximum on fifth day

Pulse rapid tongue furred constipation, bronchial catarrh

2. Stage of Nervous Excitement and of Eruption. Fifth to tenth day Characterized by restlessness rash and delirium

RASH - *Onset* Usually fourth to fifth day Commences in axilla and on wrist, then abdomen spreads to chest and extremities [illegible] on face and neck

Characters [illegible]

Subcutaneous mottling diffuse irregular and dusky

Papular spots Very irregular size and shape and feature outline Slightly elevated Pink or dirty [illegible] In early stages disappear on pressure [illegible] Usually very numerous Continue to appear for two or three days In children closer resemblance to measles owing to coalescence of spots and blotches but [illegible] appearance

DELIRIUM [illegible] towards end of fever [illegible] marked at night Often [illegible] in other cases comatose

ODOUR [illegible]

Prostration [illegible] Pulse rapid

3. Stage of Nervous Prostration [illegible]

RASH [illegible]

Other [illegible] Rapid and soft [illegible] and shrivelled Deafness

[illegible] eyes In [illegible]

In [illegible] (1) [illegible] (2) Hypostatic [illegible] (3) [illegible]

4. Crisis. [illegible] falls asleep and wakes [illegible] hours, [illegible] Relapse never

Special Features.

TEMPERATURE [illegible] slight morning remissions maximum [illegible] No fall with appearance of rash [illegible] remissions until crisis

Crisis, temperature falls to subnormal in twelve to twenty-four hours. In fatal cases rises to 108° or 109°.

LUNGS. Bronchial catarrh early. Hypostatic pneumonia later. Pulmonary complications have high mortality.

HEART.—Pulse often rapid and feeble throughout. Rarely dicrotic. Systolic murmur common. Dilatation and failure not infrequent.

URINE. Albuminuria common. Chlorides markedly diminished. Nephritis rare.

BLOOD. Leucocytosis usual.

SPLEEN. Not palpable.

Variations in Type. Mild cases occur with convalescence on tenth day. Epidemics may be very mild, e.g. Brill's disease, now known to be typhus. Malignant forms, *typhus siderans*, fatal in two to three days.

Complications and Sequelæ. Uncommon. Bronchopneumonia most frequent, is serious, may end in gangrene of lungs. Rare are nephritis, abscesses, gangrene, paralyses. May be temporary mania.

Prognosis. Almost always favorable, but varies greatly with treatment, age, and in different epidemics and surroundings. Varies directly with age. In children, 2 to 4 per cent; adults, 10 to 20 years over 15 per cent. Death most frequently occurs third week from toxemia, in third or fourth week from pulmonary infarcts.

Diagnosis. In epidemics simple. Difficulties usually in first few days before eruption. Widal reaction positive, great value.

1. TYPHOID. In typhus sudden onset, early prostration, mental symptoms with absence of diarrhea, bronchitis, tenderness and enlarged spleen, aspect dull, character of rash usually distinctive, but not invariably. *Blood culture, examination of excreta, and serum reactions* in doubtful cases. Diagnosis often very difficult.
2. MEASLES. Catarrhal symptoms, Koplik's spots. Rash begins behind ears, spreads in 1 to 2 days.
3. CEREBROSPINAL FEVER. Epidemics of spotted fever and of typhus undoubtedly have been confused. Cerebrospinal fluid distinctive.
4. PURPURA. No cerebral symptoms. Common diagnosis in ported cases.
5. ERYSIPELAS. May resemble petechiae in later stages of rash.
6. SEVERE SMALL-POX. Difficulty from initial scarlatinal rash. Eruption affects face early.
7. RELAPSING FEVER. Examination of the blood.

Treatment. In general resembles typhoid fever. Diet not so strict. Should be in open air if possible. Delirious stages need constant watching. Hydrotherapy of great assistance. Give water freely. *Drugs*, only as stimulants, especially alcohol. Other valuable stimulants are musk, gr. v., in emulsion; camphor, gr. ½, in sterile olive oil hypodermically. Retention of urine may need catheterization. *In convalescence* restrain patients' anxiety for exertion.

CHAPTER XXXIV

DENGUE.

An acute fever of hot climates due to an unknown virus conveyed by mosquitoes, and characterized by severe pains an initial fever a remission, and a terminal fever and eruption

Geographical Distribution. In tropic and subtropics only Mainly a coast disease, following trade routes

Mode of Transmission.—Note —

(a) Virus unknown Filter passes Infection follows intravenous inoculation of filtered blood from infected subject

(b) Mosquito conveys virus probably *Culex fatigans* and *Stegomyia fasciata* Occurs in epidemics affecting great percentage of population Not contagious from man to man One attack usually protects

Symptoms.

INCUBATION PERIOD Probably one to three days

INITIAL FEVER Sudden onset chill fever headache and aching of eyeballs *intense pains in joints and muscles* *Temperature* 103° to 106°, often maximum on first day *Face* rapid usual febrile symptoms *Face* suffused often swollen mucous membranes congested causing sore mouth and conjunctivitis skin erythematous, thus general condition forming so called initial eruption

PERIOD OF REMISSION Between second and fifth day (often third) temperature falls with sweating cessation of pains in joints and of headache epistaxis often occurring congestion disappears May occur by crisis or lysis rarely Duration two to three days

TERMINAL FEVER AND ERUPTION Fever and pains recur usually milder than initial stage duration twenty four to thirty six hours *Eruption* rarely absent, earliest on palms and backs of hands, later on trunk, thighs, and legs Commences as reddish erythematous areas, fading on pressure finally may coalesce but varies in different epidemics resembling measles or scarlet fever and is not characteristic in type Often persists several days *Miliary desquamation* follows

TOTAL DURATION Usually seven to eight days

CHARACTER OF PAINS Great severity *knees most constant site, also back*, but none immune Localization of pain very difficult and cause uncertain joint is not swollen and can be palpated or moved passively without discomfort but intense pain follows movement by patient nor are muscles tender though probably are cause of pain (Some observers have recorded swollen joints and hyperæsthesia absent in most epidemics)

CONVALESCENCE Protracted from mental and physical weakness Pains in one or more joints often occur intermittently for weeks

Complications.—Rare Cervical glands may be enlarged Rarely hæmorrhages, orchitis, boils

Mortality.—Direct mortality nil Debility resulting may predispose to diarrhœa, etc

Diagnosis. In epidemics simple Main symptoms are intensity of pains, period of remission, and terminal eruption Diagnosis from

INFLUENZA Occurs in cold seasons

MALARIA Not epidemic Protozoa in blood Controlled by quinine

YELLOW FEVER Slow pulse no eruption jaundice hæmorrhages

RHEUMATIC FEVER Not epidemic effect of salicylate

Various tropical fevers e.g. Seven day [illegible] In Indian coasts have some resemblance

Treatment. Symptomatic Quinine has no [illegible] hyperpyrexia hydrotherapy—Pain often [illegible] morphia Purges to be avoided if possible [illegible] motions In convalescence tonic

Prophylaxis. Anti mosquito precautions

General Characteristics. (1) Convey[illegible] (2) Virus ultramicroscopic present [illegible] (3) [illegible] periods with remission duration about [illegible] (4) [illegible] pains without arthritis (5) Terminal [illegible] (6) Mortality negligible

CHAPTER XXXI

ACUTE POLIOMYELITIS.

(Heine Medin Disease Infantile Paralysis)

An acute infection localized in the central nervous system especially in lesions of the anterior horns of the [illegible] and characterized by fever and paralyses

History. Von Heine, 1840, recognized the clinical entity Medin 1887, observed its occurrence in epidemics

Distribution.—Widespread in temperate climates throughout Europe and North America Sporadic cases common Epidemics of late, especially in Scandinavia, e.g. Sweden, 1905 and United States (New York, 1907 and 1916) General frequency increasing

SEASON Late summer and autumn greatest prevalence Epidemics diminish with cool weather

AGE Great majority *under five years* frequency diminishes

Acute Poliomyelitis—Distribution, *continued*

rapidly subsequently, but adults not immune and specially affected in some epidemics

SEX.—Both sexes

Mode of Infection.*

LANDSTEINER and POPPER (1909) transmitted disease to monkeys by intraperitoneal injection of spinal cord tissue from a fatal case

FLEXNER, (1909) results more constant by intracerebral injections

FLEXNER and LEWIS, (1911) (1) Transmission to monkeys by injection of nasal mucosa of patients, (2) Transmission from one monkey to another

PATHS OF EXPERIMENTAL TRANSMISSION (i) Intracerebral and subdural, (ii) Large nerve trunks (paralysis commences locally), (iii) Nasal mucosa, scarification and inoculation with virus or by simple injection into nasal cavity (iv) Intravenous very large injections necessary

ANIMALS SUSCEPTIBLE Anthropoid apes and lower animals

THE VIRUS

i. Filterable through earthenware filters

ii. *Distribution in tissues* Present in (a) brain and spinal cord markedly (b) Nasal mucosa (c) Lymphatic fluid (d) Symptomatic exudate (e) Ascitic fluid (f) Cerebrospinal fluid (g) Blood (h) Urine

iii. *Cultivation and artificial media* FLEXNER and NOGUCHI (1909) cultural media have been made by growth in naturally sterile serum and serum plus extract of brain of rabbits (Noguchi) Noguchi (1911) on ascitic fluid with presence of rabbit

Artificial media Minute filterable bodies (impure) or tracks in ships, than vaccine (Gram-positive) Gram, are filtered out After [illegible] on other media Sabouraud [illegible] Now demonstrated in tissue culture [illegible] experimental Nature unknown.

iv. Found in increasing proportion [illegible]

'CARRIERS' Monkeys after [illegible] nasal [illegible] infective for man, [illegible] Man chronic [illegible] recognized, even with previous mild symptoms [illegible] of disease

SERUM After infection antibodies [illegible]

i. Neutralize virus when mixed [illegible]

ii. Present in serum for many years

iii. Protect monkeys against [illegible] attack recorded in human [illegible]

iv. Injection into monkeys does not [illegible] first inoculation

v. Serum of recovered subjects injected [illegible] of early poliomyelitis is said to [illegible] paralysis Intrathecal and intravenous injection 35 to 12 [illegible]

* See Manual of [illegible]

NATURAL MODES OF TRANSMISSION — Nasal mucosa becomes infected with virus (early sore throat) spreads to central nervous system by lymphatics, of olfactory nerves and other lymphatics, and probably not by blood (Flexner). Extension in epidemics is erratic, resembling cerebrospinal meningitis, factors probably being (i) Many persons non susceptible, (ii) Healthy contacts acting as 'carriers'.

Stability, formerly suspected, now generally exonerated.

Morbid Anatomy. — Changes in the nervous system are widespread, a polioencephalitis always more extensive than clinical symptoms suggest; thus, in fatal cases with paralysis of bulb, lesions are present in bulb and often in cerebral hemispheres.

Lumbar and cervical swellings, cord most affected.

Gravest lesion — leptomeningitis, vessels distended, and infiltration of perivascular lymphatic spaces with polymorphonuclear cells spreading into cord. The primary lesions are vascular.

SPINAL CORD — *Inflammatory condition* [illegible] markedly in anterior horns of anterior commissure and extending into anterior columns; hyperæmia, cellular infiltration of perivascular lymph spaces. There may be thrombosis, rupture and hæmorrhages. *Degeneration* [illegible] of nutrition especially in anterior horn. Less [illegible] in anterior horn. Loss of small round [illegible] cells.

OLD STAGE CASES — [illegible] inflammation present. [illegible] affected [illegible] secondary degeneration.

BRAIN — Changes [illegible]

CEREBROSPINAL FLUID — [illegible] lymphocytes may be present. Very [illegible] absent.

Symptoms.—

INCUBATION PERIOD — [illegible] to ten days. May be slight malaise and [illegible] not recognized.

ONSET — Sudden. [illegible] convulsions. [illegible] less often diarrhœa, [illegible] flushed face. [illegible] Constitutional symptoms [illegible] days.

INITIAL STAGE OF PARALYSIS — Characteristics: (i) [illegible] of paralysis greatest at onset, [illegible] subsequent changes in improvement. — Asymmetry of distribution, one leg or one leg and opposite arm. Bladder and rectum rarely affected.

PAIN AND HYPERÆSTHESIA — Absent in most cases but sometimes severe.

CUTANEOUS SENSATION — Normal.

REFLEXES — Absent in affected limbs.

ELECTRICAL REACTIONS — See PROGNOSIS.

Initial stage lasts two to three weeks. By this time [illegible] pain remitting, and paralysis often already greatly improved. Reflexes may be returning.

STAGE OF RECOVERY — May continue for twelve to eighteen months. Reflexes return unless extreme wasting and reaction of degeneration present. Babinski's sign late.

DISTRIBUTION OF LESIONS — Most common is one leg; next

Acute Poliomyelitis—**Symptoms,** *continued.*

one leg and one arm. In lower extremity, extensors of hip, knee, and dorsiflexors of ankle are most affected: and in upper extremity, muscles of the shoulder. Abdominal muscles not uncommonly in young children. Marked paralysis of trunk muscles rare.

Course and Permanent Results.—During stage of recovery and as child grows, permanent results become obvious: (1) *Small size* and 'shortening' of affected limb (bones do not grow if many attached muscles are paralyzed). (2) *Deformities*, especially talipes, flexed knee, occasionally scoliosis, lordosis, mainly from action of unopposed muscles. (3) Muscles wasted. Skin usually cold.

Various Clinical Types.—Ordinary sporadic type described above. Localization of lesions in various sites occasionally produces variations. Rare except in epidemics.

ABORTIVE FORM.—Malaise, transient signs of irritation of nervous system, no paralysis. Recognized in epidemics only.

RAPID ASCENDING FORM.—Starts in legs and spreads upwards, death from respiratory paralysis in few days. Resembles Landry's paralysis, 'acute ascending myelitis.'

BULBAR FORM.—*Acute bulbar paralysis.* Paralysis of cerebral nerves (lingual, laryngeal, and pharyngeal muscles), may co-exist with spinal paralyses, may be fatal.

MENINGITIC FORM.—Resembles cerebrospinal meningitis, viz., headache, vomiting, pains in back and neck.

Acute Encephalitis.—Fever, vomiting, and convulsions occur with paralysis, hemiplegic or monoplegic, of cerebral type. It is probably the cerebral form of infection corresponding to acute poliomyelitis. Types resembling transverse myelitis and polyneuritis (with pain and hyperæsthesia) also occur.

Prognosis.

ACUTE STAGE.—Mortality low; deaths result from respiratory paralysis, either directly, or secondarily from bronchopneumonia, hence rare when these muscles are unaffected, and usually within first few days. Varies in different epidemics: in New York, 1916, was 27 per cent.

RECOVERY OF POWER.

1. Initial paralysis is the maximum; all subsequent change is improvement.
2. Improvement may continue twelve to eighteen months.
3. Improvement cannot be expected if not commencing within three months of onset.
4. *Electrical reactions.*
   i. Faradic response present. Muscles *will recover.*
   ii. Reaction of degeneration present in muscles, viz.: (a) Faradic response absent. (b) Galvanic response sluggish, with anode closure more ready than kathode closure. *Prognosis serious.* The sequel may be: (a) Gradually, complete loss of galvanic response,

denoting permanent paralysis, (b) Slow return towards normal response, showing possibility of improvement

Diagnosis.—Difficulties are mainly in infants, in cases with hyperæsthesia and in abnormal forms ordinary type usually simple

In Acute Stage diagnosis from —

1 CONDITIONS WITH PAIN IN LIMBS simulating paralysis, e.g. *scurvy rickets* (a painful swelling), *rickets* (other signs present) *rheumatic fever* (never under two years) *syphilitic* epiphysitis *osteomyelitis*

2 CEREBROSPINAL MENINGITIS (a) Cerebrospinal fluid (b) No early flaccid paralysis

3 MULTIPLE PERIPHERAL NEURITIS May be difficult Onset gradual distribution symmetrical some sensory changes

In Stage of Residual Paralysis In [illegible] reflexes are spastic in [illegible] weakness and lack growth may be present

Prophylaxis. Patients [illegible] Virus not present [illegible] except in rare carriers

Treatment

ACUTE STAGE [illegible] treatment [illegible] treatment [illegible] are [illegible] into [illegible] under [illegible]

In cases of [illegible]

PARALYZED MUSCLES [illegible] must be kept in position [illegible] allowed to be stretched actively [illegible] salt [illegible] (1) Gravity [illegible] of unopposed muscles [illegible] principle is [illegible] limb maintained at rest during acute stage of two to three weeks subsequent treatment is (1) Massage light [illegible] *movements*, patient also [illegible] galvanic current

(2) CELLULOID SPLINTS [illegible] muscles and contractures of unopposed muscles joints are necessary Also patient should be [illegible] as possible for general health and [illegible] muscles Celluloid splint have [illegible] (1) Lightness (2) Cleanliness (3) Being easily adapted to individual and frequently renewed with growth (Letter *Clinical Journal*, Nov., 1913)

SURGICAL TREATMENT Often necessary later especially tenotomies and transplantation of tendons

CHAPTER XXXVI.

ENCEPHALITIS LETHARGICA.

An acute specific disease of the nervous system, originally recognized by the syndrome of fever, lethargy, and ocular palsy, but the type has apparently changed and the symptoms are now protean.

In epidemic form, appeared in Austria in winter of 1916, in France and Great Britain in winter of 1917, and in America in winter of 1918.

Direct contagion is rare. Infection probably occurs through the nasal mucous membrane.

The virus is unknown. Strauss and Loewe report a filtrable organism resembling Flexner's 'globoid bodies' of acute anterior poliomyelitis. Transmission to animals is recorded.

Morbid Anatomy of Nervous System.—

MACROSCOPIC.—Congestion of meningeal and intracerebral capillaries. On section of brain, minute vessels distended and oozing: cortex reddish, and also gray matter of basal nuclei and mid-brain. These changes may be slight. Hæmorrhages are rare, but occasionally are large.

HISTOLOGY.—

(1) Vascular congestion.

(2) Small round-cell infiltration of the perivascular lymph spaces. This so-called 'perivascular cuffing' is the most constant change, but is often patchy in distribution. Cells are mainly small lymphocytes with a few large lymphocytes laden with pigment granules.

Other less constant or marked changes include: (3) Degeneration in the nerve-cells and neuronophagy; (4) Proliferation of the mesoblastic cells of the vessel walls; (5) Glial proliferation; (6) Venous thrombosis; (7) Hæmorrhages.

DISTRIBUTION OF LESIONS.—Mid-brain and basal ganglia most affected. Any portion of nervous system or membranes may be affected.

Symptoms. The disease was recognized by the syndrome, lethargy and diplopia (i.e., ocular palsy). The symptoms as found at present are protean, due to the widespread lesions and to the specialization of function and complexity of structure of the nervous system. Numerous 'types' have been described, but such classification is unsatisfactory, as a single case may exhibit many types. The classification given below (based on Walshe) divides the nervous symptoms into *general* and *focal*, and subdivides these groups into positive and negative. Positive symptoms denote exaltation of function, either from irritation or from loss of higher control. Negative symptoms denote depression or loss of function, from destruction of nervous tissue. The combinations of these groups are almost endless. The earlier cases were essentially of 'negative' type.

CHAPTER XXVII.

VACCINIA (*Cow Pox*) VACCINATION.

Vaccinia is an acute infective disease of cows characterized by a vesicular eruption on the udders and teats, caused by a virus which on inoculation protects man against small pox.

History.

SMALL POX. [illegible] in the Far East especially India. [illegible] Greece or Rome. In Egypt very fatal. Name can be derived from Hebrew word meaning a boil. Spread in Europe probably in 6th century A.D. [illegible] 14th century described by John of Gaddesden [illegible] and studied carefully by Sydenham.

VARIOLOUS INOCULATION. [illegible] matter [illegible], from pustules of mild cases, practised [illegible] for many centuries. Commonly in [illegible] of variola, but sometimes [illegible] by Lady Mary Wortley Montague.

Careful inoculation would certainly [illegible] death rate [illegible] of population [illegible] largely prevented [illegible] in 1840.

JENNER'S DISCOVERY OF VACCINATION.

A [illegible] protected against small pox. [illegible] matter as a prophylactic by [illegible] conceived idea of protective [illegible] about 1780. We [illegible] but rejected two forms of [illegible] which was protective, and investigated [illegible] latter similar to vaccinia. Finally, [illegible] crucial experiment by inoculation, [illegible] with matter from the hand of a girl infected with cow pox, and found that [illegible] resisted subsequent inoculation with small pox matter. [illegible] made the year following, and in 1798 Jenner published his method. [illegible] (1799) introduced [illegible] vaccination. [illegible], in America, in 1800, repeated Jenner's experiments and confirmed the resulting immunity.

Vaccination spread rapidly in all civilized countries. Opposition tended to grow after some years as vaccinated persons contracted small pox. Unfortunately, Jenner, to his death in 1823, never recognized necessity for revaccination, and tried to make excuses for every case of failure. He considered variola and vaccinia identical. His main discovery was that matter from a human being with cow-pox protected another against small-pox.

Vaccinia—History of Vaccination, *continued.*

RE-VACCINATION.—Commenced between 1820–1830, the value becoming slowly recognized.

Relation of Vaccinia to Variola.—Not fully proved whether vaccinia is an independent disease or variola modified by passage through the cow. The following experiments suggest that vaccinia was originally due to inoculation of calves with small-pox matter, and *vaccinia is generally accepted as being modified variola.* Important relations are :—

1. Cow-pox or matter from vaccinia on inoculation into man never produces variola.
2. Vaccinia is only infective when matter from it is inoculated into an abrasion of the skin.

   These two points are specially advanced by those upholding independence of the two conditions.
3. Vaccinia protects against variola and variola against vaccinia. No instance is known in which one disease protects against another.
4. Inoculation of calves with variolous matter from man never produces variola. In majority of cases no definite lesion follows in first series, but after passage through several calves definite vaccinia results. The lymph from these produces typical vaccinia, and never variola, either in children or calves. Experiments are few but carefully authenticated. Chauveau's experiments (Lyon's Commission), purporting to prove that variolous matter produced variola in calves and again on retransference to man, are not accepted now.
5. Variolous matter inoculated into monkeys, after several passages, and thence on to calves, produces vaccinia readily.

Preparation of Vaccine Lymph.—Calves are vaccinated with lymph on abdomen aseptically. The contents of the vesicles are scraped, mixed with glycerin, and stored until sterile, extraneous organisms die out in three to four weeks. Lymph remains active for two months.

Technique of Vaccination.—Cleanse skin with soap and water, and then ether. Scarify in four places over ¼ inch apart, without definite bleeding. Place drop of lymph on each and rub in with the needle. Leave to dry without covering at least a quarter of an hour. Cover with lint, and protect vesicle and pustule from rupture by gauze and strapping.

Site. - Over deltoid or above knee.

Subcutaneous injections are under trial.

Symptoms of Normal Vaccination.—

LOCAL SYMPTOMS:—

Third day : *Papule* with red zone.

Sixth day : *Vesicle* with umbilication, red zone increases.

Eighth day: Vesicle of maximum distention, marked umbilication.
Tenth day: *Pustule.* Skin swollen and painful.
Twelfth day: Pustule commences to dry; hyperæmia diminishes.

Scab separates about twenty-first day and leaves scar.

CONSTITUTIONAL SYMPTOMS.—Variable in degree: malaise and restlessness may be considerable. Pyrexia, usually slight, third to eighth day with eruption. Axillary (or inguinal) glands palpable. Definite leucocytosis.

Abnormalities of Vaccination.—

1. Local vesicles may form round primary zone.
2. Transient rashes, erythema or urticaria, in second week, rarely purpura.
3. Inflammations and deep ulcers may result from uncleanliness and secondary infections, or from injury: usually weak subjects.
4. *Generalized Vaccinia.* Very rare. A general pustular eruption, usually commencing on eighth to tenth day: formation of pustules may continue for several weeks. In children occasionally fatal.

All severe complications are extremely rare.

Postpone vaccination of children who have specific or skin diseases or any marked ailment.

Transmission of Disease by Vaccination.—

SYPHILIS. Transmission by vaccination was possible in arm-to-arm inoculations, but not with modern calf lymph.

TETANUS. Cases have occurred.

TUBERCULOSIS. No undoubted case is known.

Vaccination and Re-vaccination.—Vaccination should be performed between fourth and sixth months. Re-vaccination at 9 years, and again at 21 years, *and always after exposure to infection.* *Complete vaccination* should show four scars from first vaccination and also scars from re-vaccination.

Lesions and symptoms in re-vaccination may be identical with primary vaccination, especially after long intervals; in other cases course is shorter and less severe, and all degrees occur to complete absence of reaction.

Duration of Immunity and Value of Vaccination.—

Immunity is complete three weeks from vaccination.

After exposure to infection, *immediate vaccination* protects completely, or will greatly modify course of variola: vaccination later in the incubation period will modify course if eruption of vaccinia appears two or three days before variola commences.

Degree of protection varies with number of scars. With complete vaccination and re-vaccination mortality is nil. With four marks case-mortality is 2 to 3 per cent and case-incidence low.

(*See also* VARIOLOID, p. 206.)

Duration of Protection.—Probably ten to fifteen years.

CHAPTER XXVIII.

CHICKEN-POX.

(*Varicella.*)

An acute infectious disease, due to an unknown virus, and characterized by a vesicular eruption usually appearing in successive crops. Rarely severe.

Etiology.—Endemic and sporadic, occasionally epidemic.

AGE.—Usually under 10 years. Infants may be attacked. Adults very liable, if no attack in childhood.

SEASONAL VARIATIONS. - Slight.

RELATION TO VARIOLA.—Entirely distinct. Note:—

1. No mutual immunity conferred by an attack of either.
2. Patient with varicella can be vaccinated.
3. No cases of variola occur during epidemic of varicella.

'Varioloid varicella' occurring in the West Indies is a mild form of variola.

Morbid Anatomy.—The formation of a pock commences in the middle layer of the prickle cells. The nuclei divide, and the cytoplasm becomes swollen and vacuolated, degenerates, and liquefies. Lymph is exuded.

Mode of Infection.—*Highly contagious.* By direct contact, by infected articles, by third persons, or by the air over short distances. Cannot be inoculated by blood. Nothing is known of the virus.

One attack usually, but not invariably, protects. *Insusceptibility* probably not uncommon.

DURATION OF INFECTIVITY.—Until all crusts have separated without re-forming, usually about one month. Delay frequently caused by one or two obstinate pocks.

QUARANTINE PERIOD FOR CONTACTS.—Three weeks.

INCUBATION PERIOD.—Variable: ten to seventeen days, commonly fourteen days. Limits: ten to twenty-one days.

Symptoms.—

STAGE OF INVASION.—In *children*, usually slight fretfulness and anorexia. In *adults*, pyrexia, slight chill, vomiting, pains in back, usually slight, rarely severe and suggestive of small-pox. *Prodromal rash*, a general erythema, occasionally occurs. Initial symptoms often overlooked until eruption attracts attention.

ERUPTION.—On first or second day. Fever does not disappear with eruption, but symptoms are slight throughout.

ORDER OF APPEARANCE OF ERUPTION.—*Earliest on trunk*, either back or chest. Rarely on forehead or limbs. Few spots in mouth at same time. No constant sequence subsequently.

DISTRIBUTION OF FULL ERUPTION.—Usually characteristic: (a) Trunk and scalp most affected; (b) Face and limbs less

so, and proximal portions more affected than distal. Few spots on palms and soles, often none. May occur on palate. Sometimes on labia and in urethra. On scalp, hands, and feet the vesicles are small and may be 'shotty'.

CHARACTER OF ERUPTION.—Rose-coloured *papules*, changing in few hours into *vesicles*, size of match-head. Contain clear serum. No umbilication. Firm, but more superficial than, and without shotty feel of, small pox. Always discrete. Skin around normal, or slight red areolæ. *Pustules* form in forty-eight hours: later shrivel and form crusts.

SUCCESSIVE CROPS. On subsequent days. Usually three in all.

ALL STAGES OF ERUPTION SIMULTANEOUSLY PRESENT, even among those of similar date.

NUMBER OF SPOTS.—Ten to several hundreds.

PROGRESS OF ERUPTION.—The progress of different spots usually varies; some vesicles may not become pustular.—

1. Pustule remains unruptured, falls off in five days to two or, rarely, three weeks, leaving dry surface, no scar.
2. Pustule ruptures from scratching or injury. Thin crust forms, dries rapidly, scab falls in one to three weeks.
3. Pustule ruptures, skin around becomes inflamed, crust forms with suppuration below, falls off in one to two weeks. Surface ulcerated but heals rapidly. *Scar* often results. More common in children, especially on face.

Constitutional Symptoms.—General disturbance depends on number of spots, amount of pustulation, and ulceration. *Itching* may be severe and cause insomnia. *Temperature* 99 to 101°, occasionally 103°, but rarely longer than three to four days. Often rises with each crop. Falls rapidly. May rise again in second week with suppuration under crusts. *Constitutional symptoms* rarely severe, even with the higher temperatures, except in debilitated subjects. In adults both eruption and constitutional symptoms often severer.

Variations in Eruption.—

NECROSIS AND ULCERATION. Not uncommon in uncleanliness. General symptoms severe, varying with degree of ulceration. Is cause of most fatal cases. *Gangrene* round vesicles rare.

VARICELLA BULLOSA. Rare. Large bullæ develop from the vesicles. Severe itching and general symptoms.

HÆMORRHAGIC VARICELLA. Very rare. Recoveries occur.

Duration. Acute stage three to seven days, depending on number of crops; rarely twelve days. (*See* DURATION OF INFECTIVITY.)

Diagnosis.—Usually simple. Characteristics are: (*a*) Order of onset of rash; (*b*) Distribution; (*c*) Successive crops; (*d*) Various stages of eruption simultaneously present: papules, vesicles, and pustules; (*e*) Symptoms slight, but temperature does not fall with appearance of rash.

VARIOLA.—*See* SMALL-POX, p. 207.

Chicken-pox—Diagnosis, *continued*

IMPETIGO CONTAGIOSA.—Mostly on face. Mucous membranes not affected.

HERPES ZOSTER.—Definite distribution corresponding to nerve roots. Some evidence exists that virus is the same.

Treatment.—For mild cases no special treatment. *Itching*: sponge with warm boracic lotion, or dust with starch and zinc oxide powder. Prevent scratching. Cut hair if much eruption on scalp. For ulceration, apply hot fomentations. Warm bath hastens separation of scabs, but these re form if separated too early.

CHAPTER XXIX.

SCARLET FEVER.

(*Scarlatina.*)

An acute infectious disease of unknown origin characterized by inflammation of the fauces and a punctate erythematous rash followed by desquamation, and by a special tendency to nephritis and otitis media.

There is much uncertainty as to factors and duration of infectivity.

Etiology.—

GEOGRAPHICAL DISTRIBUTION.—In all temperate climates. Endemic and frequently epidemic. Uncommon in tropics.

SEASON.—Marked seasonal prevalence. Increases during summer to maximum in October, rapid fall in December, minimum in March. Slight fall in August due to closure of schools.

AGE.—Most frequent about five years of age. Over 80 per cent under ten years. Frequency diminishes in each subsequent decade.

VIRULENCE.—Varies considerably in different epidemics and years. Present mortality 1 to 3 per cent, highest in first years of age. Epidemics of malignant type may occur. Second attacks not so universal as in measles.

One attack usually protects for life.

Morbid Anatomy.—Nothing characteristic apart from kidneys. *Rash* not visible post mortem unless haemorrhagic. Fauces: acute inflammation. *Cervical lymphatic glands* may be enlarged. General changes of acute fever, but spleen not enlarged. Endocarditis not uncommon, rarely pericarditis. Pulmonary complications frequent in fatal cases.

RENAL CHANGES.—*Nephritis* not uncommon; changes usually not characteristic, but occasionally a pure glomerular nephritis.

SPECIFIC VIRUS.—Unknown. Very resistant.

The relationship of streptococci has been much disputed. Note:

1. Streptococci are very frequently present in fauces, pus, etc., in scarlet fever.

2. The strain usually isolated differs from *Str. pyogenes* in certain particulars, e.g., clots milk, forms conglomerate masses in peptone broth (Gordon's *Str. scarlatinæ*)
3. Undoubted streptococcal infections may produce rashes and faucial inflammation indistinguishable from scarlet fever. Outbreaks of scarlet fever do not arise from these, though infection may spread with similar symptoms to 'contacts' with wounds.

It is generally believed that scarlet fever is not due to streptococci, though these are responsible to some extent for the complications.

- *Experimental Inoculation.*—Infection is recorded by subcutaneous inoculation of mucus from the mouth into human beings. Said to have been transmitted to monkeys.

Among animals, there is no evidence of its occurrence naturally.

Mode of Infection.—

CONVEYANCE OF INFECTION.—(i) *Direct contact* with infected persons, usual cause. (ii) *Infected articles* may be conveyed by clothes, books, etc., for long distances and time, certainly several months. (iii) *Third persons.* (iv) *Milk borne epidemics.* Milk may be infected in transit by individual with scarlet fever. Some epidemics have been ascribed to a certain pustular eruption of cows' udders, e.g., in 1885 (Klein), and 1909 (Hamer and Jone[illegible]); the evidence is not fully convincing.

Aerial conveyance negligible; no cases occur near fever hospitals. *Water* of no importance.

INFECTIVE MATERIAL FROM PATIENTS.—(a) *Secretion of throat, nose, and ear.* Undoubtedly main cause of infection. Infectivity probably persists as long as nasal secretion is abnormal. (b) *From skin.* Generally considered infectious during rash. Infectivity from scales during desquamation is doubtful. After fourth week, evidence exists that scales are not infectious. No proof exists that scales are infectious at any time, but possibility is not disproved.

Infectivity of urine is unknown.

'RETURN CASES.'—In 3 per cent. of cases discharged subsequent infection of the household occurs usually within two weeks of discharge. Undoubtedly not connected with peeling, but cause not fully known. Often original case has nasal discharge or otorrhœa or faucial inflammation after leaving hospital. It is possible (1) 'Carriers' exist for a short period, probably connected with nasal or other discharges. Simple coryza may cause these discharges to become actively infective. (2) Re-infection occurs from patients in earlier stages: no evidence for this, and contrary to rarity of relapse.

DURATION OF INFECTIVITY.—General principles.—

1. *Isolation should be complete for six weeks* from development of rash, and up to twelve weeks if attack severe.
2. It is unnecessary to isolate until desquamation is complete.
3. No case should be regarded as free from infection in which

Scarlet Fever.—Mode of Infection, *continued.*

nasal or aural discharge is present. If these persist isolation must be complete for minimum of twelve weeks, and preferably up to twenty weeks.

QUARANTINE PERIOD FOR CONTACTS. Seven days.

INCUBATION PERIOD. Generally two to four days, most commonly three days. Limits half to six days.

Clinical Varieties.

Four types: (1) Simple, ordinary form, scarlatina benigna. (2) Malignant or toxic. (3) Hæmorrhagic. (4) Septic or anginose. Intermediate types occur. All severe forms are rare.

SYMPTOMS

1. Simple Scarlet Fever (*scarlatina benigna*). Three stages: (1) Invasion. (2) Eruption. (3) Desquamation.

1. STAGE OF INVASION.—

Onset. Sudden. Chilly sensations, almost rigors, infrequent. Convulsions not uncommon in children, also epistaxis.

Initial Symptoms. (a) *Sore throat*, with some tenderness on swallowing or in submaxillary region. (b) *Vomiting*, early and constant. Sore throat is commoner in adults and vomiting in children. (c) *Temperature* rises rapidly, often 103° to 104° when first taken. (d) *Pulse* very rapid, especially in children. Skin dry and very pungent. *Face* flushed. *Tongue* furred. General malaise and constipation. No definite diagnosis until rash appears. In early signs mild attack, mild at least; severe attack may still be mild.

2. STAGE OF ERUPTION. *Rash* commences twenty-four to thirty-six hours after onset, usually on second or third day, occasionally more rapid, rarely delayed for three to four days. General *aggravation of symptoms*: throat more swollen and painful, tongue more furred, temperature higher, and pulse more rapid. Symptoms increase for two to three days, then simultaneously, rash fades, defervescence occurs, and symptoms abate. *Convalescence* usually on sixth to eighth day.

3. STAGE OF DESQUAMATION. Skin rash subsides, leaves stained and rough. *Desquamation* or peeling commences on the neck, follows order of rash, *and occurs last on palms and soles*. May commence *before* rash has faded on limbs. Extent proportional to rash. On the face it begins at numerous foci and separates as powder, on abdomen as scales, on soles of feet as large flakes. Most marked in second week, usually complete in four weeks except soles. May be many weeks. Usually slight in infants. Slight secondary desquamation is common. Nails subsequently have transverse ridges more frequently than in any other fever.

Special Features.—

RASH. *Onset on second or third day.*

DISTRIBUTION.—*Commences on neck, behind ears and angles*

part of chest; spreads over body, usually in a few hours; may take two or three days.

Chest and neck, flexor surface of elbow and inner aspect of thighs most affected

Face, scalp, palms, and soles very rarely affected

CHARACTER A vivid, scarlet, punctate erythema, composed of two factors (a) scattered red spots, on (b) basis of general erythema Disappears on pressure, unless petechial

Skin smooth at first, then rough. Swelling and inflammatory œdema not infrequent, especially on hands *Miliary sudamina* or even vesicles may be present *Petechiæ* not uncommon, especially in fold and creases of skin and on neck Itching varies rarely excessive On roof of mouth and on cheeks may be a punctate eruption *Rash on extremities* sometimes blotchy and macular

DURATION Usually two to three days darkens in colour and fades roughly in order of appearance, last from sites where thickest. Generally absent by seventh to eighth day. Petechiæ may persist longer When rash subsides, may be [illegible] red line at bend of elbow, often persisting long

FACIAL ASPECT *Cheeks flushed* while mouth and nose are pale, so called 'circumoral pallor', often very suggestive Flush, peach blossom tinge of cheeks as if rouged

FEVER High at onset 103 to 104°, maximum on third or fourth day slight morning recession Declines with fading of rash Normal in one week

PULSE Rapid, out of proportion to temperature usually 120 to 150.

TONGUE In stage of invasion is furred in centre with red papillæ projecting and red at edges the 'strawberry tongue' Fur clears on third or fourth day, leaving surface red and raw, 'raspberry tongue'

FAUCES The changes may be (a) Slight redness and swelling; (b) Follicular tonsillitis (c) Membranous angina great tenderness and induration in neck and swelling of glands.

CERVICAL LYMPHATIC GLANDS Palpable

SKIN Hot and extremely pungent

These symptoms with the early appearance of vomiting and the subsequent desquamation are characteristics of scarlet fever

RHINORRHŒA. Mucous discharge common.

LEUCOCYTOSIS.—Present

URINE.—Febrile changes with early albuminuria.

GASTRIC DISTURBANCE.—Uncommon after initial vomiting.

SPLEEN.—Rarely palpable

WASSERMANN REACTION. May be positive for a short period about the third day.

Progress of Symptoms.—

First Day.—Sore throat, vomiting, high temperature.

Second Day.—Rash, strawberry tongue, rapid pulse.

Fourth Day.—Raspberry tongue.

Scarlet Fever—Progress of Symptoms, *continued.*

Fifth Day.—Rash, temperature, and symptoms commence to decline.
Sixth to Eighth Day.—Temperature normal, symptoms subside, desquamation commences.

2. **Malignant or Toxic Scarlet Fever.**—Characterized by slightness of throat lesions with severe constitutional symptoms. *Onset* severe serious vomiting, high temperature, and delirium Fauces little changed. *Rash dusky*, or may be absent.
   Subsequently: dyspnœa, rapid pulse, hyperpyrexia, coma, and cardiac failure Is a toxæmia.
   Adults occasionally recover, children never *Death* in one to two days, rarely longer.

3. **Hæmorrhagic Scarlet Fever.**—Very rare Hæmorrhages into skin and from mucous membranes, including epistaxis and hæmaturia. Invariably fatal, usually second or third day.

4. **Septic or Anginose Scarlet Fever.**—Characterized by ulceration and necrosis, commencing in fauces and spreading widely.
   CONSTITUTIONAL SYMPTOMS - Severe from onset, but septic condition prominent from third or fourth day
   FAUCES -*Lesions severe*: tonsils and palate extremely swollen, extensive exudation or membrane formation *Necrosis* commences at junction of uvula and tonsil Sloughing may be extensive and fatal hæmorrhage may occur
   NASAL DISCHARGE —Mucopurulent
   CERVICAL GLANDS - Enlarged and matted together cellulitis of neck may develop
   RASH.—Generally marked, often delayed to the fifth day usually dusky, blotchy on extremities
   PROGRESS Improvement may commence after two weeks In other cases, ulceration and necrosis extend irregularly Palate may perforate. Stomatitis severe. Aspiration pneumonia may develop. Temperature high 104° to 106° Constitutional symptoms severe rapid wasting, marked prostration cardiac failure *Death* in second or third week If patient lives into fourth week, sloughing of glands of neck (commonly), otitis media, or boils may occur and be fatal 'Secondary rashes' of septic origin not uncommon in third week
   PROGNOSIS Mortality is high, but many cases recover Wasting and weakness are always extreme, and convalescence prolonged
   Note Syphilis may be imitated by perforation of the palate also stomatitis on corners of mouth may result in radiating scars

ATYPICAL VARIETIES.

Mild and Abortive Forms. -
   MILD —All symptoms very mild, brief, and easily overlooked
   ABORTIVE.—Some characteristic symptom absent, especially rash (*scarlatina sine eruptione*) Nephritis may follow, possibly desquamation. Occurs in epidemics, and among nurses in fever hospitals.

Surgical Scarlet Fever.—Symptoms indistinguishable from scarlet fever, but always mild. Liability to follow burns and scalds definite: more rarely in operation and other wounds.

Is this syndrome true scarlet fever or of septic origin? Much disputed. Following may be stated: Patients with wounds, in contact or contiguity, may be infected and reproduce similar condition, and hence case should be isolated from them. In other contacts, e.g., nurses and medical patients, tendency to infection and the symptoms resulting agree with infectivity in other acute septic conditions, e.g., of tonsils and fauces.

Puerperal Scarlet Fever.—The relation between scarlet fever, puerperium, and puerperal septicæmia has been much discussed. Following may be stated: (1) A woman has no increased tendency to contract scarlet fever in the puerperium. (2) When scarlet fever is contracted in the puerperium: (*a*) If retained placenta or other septic condition be present, the tendency to septicæmia, and its severity when occurring, are probably increased; (*b*) In absence of retained placenta, etc., there is no increased tendency to puerperal septicæmia. (3) Septic complications of scarlet fever must be treated with special care.

IMPORTANT COMPLICATIONS.

1. **Renal** (*see also* MORBID ANATOMY)—

INITIAL ALBUMINURIA. Of febrile origin, not uncommon while temperature high, disappears when temperature falls: unconnected with subsequent nephritis. No subsequent symptoms.

NEPHRITIS. Occurs in about 5 per cent of cases; varies in different epidemics. *Onset* usually towards end of third week: may be later. No age exempt, but commoner in children. May occur even in mild cases, but especially in septic forms.

SYMPTOMS.—All grades of severity, from ample albuminuria and perhaps slight œdema, to subacute or acute nephritis with definite symptoms, and blood, casts, and much albumin in urine.

TERMINATION.—

a. Recovery, in great majority. If urine becomes free from albumin, kidney may be regarded as entirely recovered. Duration of attack usually about four weeks, but with relapses may be several months.

b. Chronic nephritis. Rare.

c. Uræmia. Very rare. Nephritis always acute.

2. **Otitis Media.**—Especially in children: rare after age of 15. *Onset* any time after first week, rarely earlier. Is due to extension of inflammation from fauces. In septic forms and angina almost invariable, but also common in mild attacks. May be usual symptoms of earache, etc., but more frequently no pain until otorrhœa occurs. Often bilateral.

Scarlet Fever—Complications, *continued*

PROGRESS.—Usually good, *with efficient treatment*, discharge ceasing in two to four weeks, and in these cases hearing not greatly affected. Complete deafness from labyrinthitis rare. *Mastoid abscess* develops occasionally: mortality high owing to sequelæ of thrombosis of lateral sinus, pyæmia, cerebral abscess, and meningitis.

3. **Arthritis.**—(i) *Multiple arthritis* (rheumatism) very frequent in adults, uncommon in children. Commences at end of first week. Small joints mainly affected. Changes in joints slight or absent. Usually, but not invariably, reacts to salicylates. Prognosis good. Chorea and cardiac affections rare. Probably unconnected with acute rheumatic fever. (ii) *Pyæmic suppuration* of joints rare, but usually fatal.

4. **Adenitis.**—Practically constant. (i) In simple and mild types, submaxillary glands tender and swollen. (ii) In anginose and septic types extreme swelling of glands, with cellulitis or subsequent sloughing. An adenitis may occur in third week, especially with nephritis. Rarely a suppurative adenitis develops in the fourth week. *Retropharyngeal abscess* occasionally occurs during convalescence.

5. **Diphtheria.** Occasionally occurs in convalescence, usually fourth week. Commonly no membrane present. Tonsillitis or nasal discharge commencing late should be examined bacteriologically.

6. **Cardiac Complications.** (i) Sudden death during convalescence, usually no previous warning, very rare. (ii) Endocarditis rare. (iii) Malignant endocarditis or purulent pericarditis in septic type.

7. **Rhinitis.** Very frequent. Nasal discharge at first thin and irritant, later muco-purulent, often obstinate, undoubtedly infective.

8. **Bronchitis.**—Common in children, usually present in fatal cases.

Rare Complications.

Chorea, Acute Poliomyelitis and Various Paralyses, Stomatitis, Noma, Perforation of Palate.

Typhoid Form.—Temperature sometimes persists for several weeks, and a typhoid state develops and may be fatal. Initial throat symptoms often slight. Usually due to a septic focus. Cervical adenitis may be present. Occasionally no cause found.

Secondary Rashes.—Usually severe cases, generally second or third week. Many types occur, e.g. (*a*) Scarlatiniform, especially on trunk and extremities. (*b*) Septic rash, mainly on extremities: irregular, blotchy, papular, or macular eruption.

Relapses.—True relapses occur, but are very rare.

Association with Other Diseases. Not uncommon: with diphtheria in 2 per cent, chicken-pox 2 per cent, measles 1 to 2 per cent of cases.

DIAGNOSIS.

Often simple, especially in cases of moderate severity: in mild forms, often very difficult: in the rare very acute forms, diagnosis may depend on existence of an epidemic, or knowledge of exposure to infection. Diagnosis never certain previous to eruption.

Differential Diagnosis.—*Difficulties in diagnosis* arise from: (1) Inflammations of the fauces: (*a*) follicular and catarrhal tonsillitis, (*b*) diphtheria. (2) Various eruptions, measles, rubella; small pox, initial rashes; erythemata of various types.

ACUTE TONSILLITIS. *Catarrhal tonsillitis* identical with throat of scarlet fever, and early diagnosis usually impossible, but tongue remains furred, rash absent or pure erythema, and no subsequent desquamation. *Follicular tonsillitis* is not so common in scarlet fever, but in diagnosis this is only of slight assistance.

DIPHTHERIA. May co-exist. Diagnosis often impossible except by bacteriological examination. Usually in diphtheria: (i) Temperature is lower and pulse less rapid, (ii) Aspect gray; (iii) Vomiting not so common, (iv) Albuminuria early, (v) Strawberry tongue uncommon.

In scarlet fever, membranous angina rarely spreads to larynx, but is more extensive on fauces, while glands are larger.

MEASLES.—In early stages, note: (i) Longer prodromal period; (ii) Marked catarrhal symptoms and conjunctivitis, (iii) Sore throat less prominent, (iv) Koplik spots, (v) Vomiting less common. In eruptive stage: (i) Macular rash, (ii) Rash present on face, (iii) No circumoral pallor. Desquamation only branny, no leucocytosis. In anginose scarlet fever, rash on extremities is often macular.

RUBELLA. Diagnosis from mild scarlet fever often difficult. Note: (i) Short prodromal period, (ii) Constitutional symptoms slight, no vomiting, no 'strawberry tongue', (iii) Sore throat slight, (iv) Occipital glands enlarged, (v) Rash occurs on face and often palate. Though frequently confluent on trunk, rash is usually discrete on lower extremities.

SMALL-POX. Prodromal rash often scarlatiniform in character, but usually localized distribution. Rash transient, initial rigor and symptoms, true eruption by fourth day, no sore throat.

ERYTHEMATA.—The rash in erythemata of various origins may resemble scarlet fever more or less closely. The history and other symptoms usually indicate the diagnosis. The most important are:—

1. DRUG RASHES. Especially belladonna, quinine, and salicylates. Iodide and bromide rashes are usually pustular.
2. ANTITOXIN RASHES.
3. SEPTIC RASHES.
4. ENEMA RASH.—Generally within few hours of enema, and usually confined to trunk.
5. FLANNEL RASHES.
6. ACUTE EXFOLIATIVE DERMATITIS.—Rare. Rash and even

Scarlet Fever—**Diagnosis,** *continued*

symptoms may resemble scarlet fever at onset. Rash more persistent, may be several weeks. Tendency to relapses and recurrences. Eosinophilia may be present.

Diagnosis in Post-febrile Stage.—Rash may linger on outer surface of legs. Peeling latest on palms and soles. Transverse lines at elbows, and cervical glands, may assist.

PROGNOSIS.

AGE.—Highest mortality in infants under one year. Greatest number of deaths occur at about 5 years. Mortality subsequently very low, and diminishes with increasing age.

GENERAL MORTALITY.—Not exceeding 3 per cent., usually lower, but varies in different epidemics.

CLINICAL TYPES.—*Malignant and anginose* types have highest mortality, usually cardiac failure either in first few days or later stages. *Ordinary types*, deaths almost confined to complications.

COMPLICATIONS. *Diphtheria*, considerable mortality. *Otitis media*, prognosis good except with sequelæ, when mortality is high. *Nephritis*, prognosis good when recognized and treated, especially in adults; when overlooked or initial scarlet fever missed, mortality higher; chronic nephritis rarely develops, serious if previous nephritis existed.

SERIOUS SYMPTOMS. *Bronchitis* in children. Severe vomiting. Hyperpyrexia, very rapid pulse, delirium. Excessive œdema or exudation on fauces. Rapid emaciation in later stages.

TREATMENT.

Course cannot be cut short, but treatment of symptoms and complications is of great importance.

Prophylaxis (*see* MODE OF INFECTION, p. 215). Patients must be isolated completely. Careful sterilization of hands by attendants.

General Hygiene. Isolation in bed. Carpets, etc., removed. Temperature constant, not over 60. Air dry, free ventilation. Light clothing. Tepid sponge daily. Encourage drinks of simple lemonade. Morning saline aperient. During desquamation rub with carbolized vaseline. Test urine daily for albumin, especially in second or third weeks. Sit up when temperature normal one week. Out of bed three weeks from onset. Out of doors a few days later, but avoid chills.

Diet.—In febrile stage, milk, beaten-up eggs and custard. When temperature normal add bread and butter, milk puddings, fruit. After three weeks, with no albuminuria, boiled fish; pass rapidly to full diet. With much faucial tenderness semi-solids easier than fluids, e.g., custards and jelly. Meat juice also useful temporarily.

Faucial Lesions.—Must always be treated locally. In mild cases spray with antiseptics, e.g., listerine, hydrogen peroxide, saturated solution of boracic acid, or chlorine water. Fauces may also be swabbed with these solutions.

In severe cases, especially septic type, syringe with Higginson's syringe or from douche-can with above solutions. After improvement, swab with carbolic acid, 1–60.

For ulceration of larynx, use bronchitis kettle with steam tent. Tracheotomy if marked dyspnœa: never intubation.

Local treatment as above 3-hourly to three times a day.

Cervical Adenitis.—Ice better than hot fomentations. Do not incise too early.

Nasal Passages.—In septic cases or with rhinorrhœa or adenoids, syringe nasal passages gently with warm water or salt water. Prevent nose-picking. Treatment probably protects the ear.

Nephritis. Protective measures include milk diet for three weeks: return to this if albuminuria occurs subsequently. Treatment as for other forms of nephritis.

Otitis Media.—For earache, hot fomentations over the whole ear. Pour a few drops of warm laudanum into external meatus. If severe, leeches behind ear. Watch drum, puncture if bulging.

For otorrhœa: treatment by usual methods.

Arthritis.—Wrap limbs in cotton-wool. Salicylates usually control pain. Practically never serious.

Hyperpyrexia and Delirium. Especially occurs in septic type. Treat by hydrotherapy. Above 103 tepid sponge. If still rises, warm bath, commencing at 90°, cooling to 80°. If delirium present, cold packs preferable. Antipyretic drugs useless.

Malignant Form.—Give stimulants, especially brandy and champagne. Wet packing or baths.

Diphtheria.—May be present. In doubtful cases give antitoxin without waiting for bacteriological results.

Convalescence.—Post-scarlatinal anæmia is frequent, especially with nephritis. In all cases give iron tonic.

Serum and Vaccine Therapy. Serum treatment is valueless. Autogenous vaccines should be used in septic cases.

CHAPTER XXX.

MEASLES.

(*Morbilli.*)

An acute infectious disease of unknown origin, and extreme contagiousness, characterized by coryza, a skin eruption, and catarrh of the upper portion of the respiratory tract.

Etiology.—

CLIMATE.—Endemic and epidemic in temperate regions, but no zone exempt.

SEASON.—In British Isles, maxima in December and June.

Measles—Etiology, *continued.*

Susceptibility universal. *Most contagious* of all fevers.

No age immune, but in civilized countries few escape past childhood.

One attack protects: second attacks almost invariably include an error in diagnosis.

Morbid Anatomy.—Nothing characteristic. *Bronchopneumonia* almost invariable in fatal cases. *Tuberculosis* is a common sequel.

Mode of Infection.—Specific virus unknown. Present in secretion of nose, mouth, and respiratory tract: and apparently in blood and skin. *Transmitted* by direct contact. Possibly, but not indisputably, by third persons, clothes, etc, but, at most, only for short distance and time. Never milk- or water-borne.

'CARRIERS'.—No knowledge, positive or negative.

EXPERIMENTAL INOCULATION.—Transmitted to man by inoculation of blood (Hektoen): also to Rhesus monkeys by blood and mucous secretions, but not by epithelium (Anderson)

DURATION OF INFECTIVITY.—*Contagiousness* is especially marked in catarrhal pre-eruptive stage, probably greatest on first day of prodromal symptoms. Very slight after rash fades, and may be considered *absent three weeks from onset of rash*, except with pulmonary and other complications

QUARANTINE PERIOD. For contacts, three weeks: must then be no catarrh or fever.

Symptoms.—

INCUBATION PERIOD (to onset of prodromal symptoms).—Usually nine to fourteen days. Limits, seven to twenty-one days.

PRODROMAL STAGE.—*Period of invasion and catarrhal symptoms.* Duration usually four days. Extremes three to six days.

ONSET OF SYMPTOMS.—Usually abrupt, but may be insidious. (1) Coryza with sneezing and thin nasal discharge; (2) Redness of the conjunctiva and lids, lachrymation, often photophobia. (3) Pyrexia moderate, commonly 102°. Cough and voice hoarse, tongue furred. Patient thirsty, restless, and irritable.

ON SECOND AND THIRD DAY.—*Face becomes puffy*: coryza, bronchitis, and conjunctivitis increase, and appearance becomes suggestive. *Koplik's spots* now appear.

Mucous membrane of mouth and throat hyperæmic and dry.

Laryngitis is common.

Temperature commonly falls. A distinct remission of symptoms may occur and be deceptive.

Glands behind jaw frequently palpable. In severe cases, convulsions, headache, nausea, or vomiting. Occasionally epistaxis.

PRODROMAL RASHES.—Occasionally on first or second day: (1) Erythema resembling scarlet fever, most common: usually on trunk only. (2) Blotchy erythema resembling true rash of measles.

MODES OF ONSET.

1. INSIDIOUS TYPE.—Gradual onset, with or without prodromata (*see* INITIAL AND GENERAL SYMPTOMS). Tendency to develop 'negative' types of symptoms—e.g., lethargy, paresis.

2. ACUTE TYPE.—Sudden onset, with or, not uncommonly, without prodromata. Symptoms usually are distinctly of the 'positive' type—e.g., acute delirium. Common predominant symptoms at onset are: (1) Psychoses—mania, etc.; (2) Convulsions; (3) Severe neuralgic pains.

3. MILD ABORTIVE, OR INFLUENZAL TYPES, OR FORMES FRUSTES.—The general symptoms occur in a mild form, suggesting influenza, and not progressing to any nervous symptoms. Diagnosis depends on the occurrence, several weeks later, of various sequelæ, especially involuntary movements and tremors, these being common with a mild initial attack; occasionally the initial attack may be entirely absent.

INITIAL AND GENERAL SYMPTOMS.—Include weakness, headache (often occipital), vertigo, shivering, muscular pains, vomiting, other gastro-intestinal disturbances, rapid pulse, and occasionally cutaneous eruptions. *Temperature* variable: may be 102°-103°, rarely more than few days; or may be absent, especially at onset, and then rise later. Severity of these symptoms is variable: not closely related to degree of nervous system involvement.

The duration of the prodromal period is usually 1 to 7 days, but may be 1 to 3 weeks.

With the progress of the symptoms, the 'Parkinsonian mask' develops, a mask-like expressionless facies, the most common characteristic symptom of the disease.

NERVOUS SYMPTOMS—

1. GENERAL.

i. *Positive.*—Delirium, mania, restlessness, etc. Certain more special types: (*a*) Acute delirium; (*b*) Confusional delirium (as in enteric); (*c*) Restlessness or insomnia at onset, progressing to lethargy (myoclonic movements common); (*d*) Korsakow's syndrome (rare). Rarely, dementia præcox, etc.

ii. *Negative.*—Lethargy, all grades from apathy to coma. Frequently somnolence by day and insomnia by night. Two special phenomena perhaps belong to this group: (*a*) Catalepsy, (*b*) Parkinsonian mask.

2. FOCAL.

i. *Positive.*—(*a*) Muscular pains (probably of thalamic origin); (*b*) Rigidity; (*c*) Involuntary movements; (*d*) Ataxia; (*e*) Convulsions (may be general).

Note on involuntary movements.—(1) Parkinsonian type: with mask, general rigidity, and tremor, but differs from paralysis agitans in abrupt onset. (2) Choreiform and athetoid movements: often in

Encephalitis Lethargica—Symptoms, *continued.*

late convalescence. (3) Myoclonic type: rapid rhythmic contraction of muscles, 30 to 40 to minute, especially affecting abdomen and diaphragm, but may affect entire musculature or a group of muscles or part of a single muscle. Onset may be two to three months after attack, often of mild character; but in some cases occurs in acute stage, the attack often commencing with insomnia and lancinating neuralgic pains, progressing to acute delirium, then developing movements, and terminating with lethargy and palsies.

ii. *Negative.*—Represented by paralyses. Ocular palsies most common, but no portion of the nervous system is immune: e.g., facial palsy, aphasia, bulbar palsy, monoplegias, diplegia, peripheral neuritis.

Note on ocular manifestations (lesions of gray matter of mid-brain).—(1) Third nerve palsies, most common, viz., diplopia, ptosis, paralysis of accommodation. (2) Pupil reactions: commonest, react to light but not to accommodation: occasionally, no reaction, rarely, Argyll Robertson reaction. Pupils may be unequal. (3) Nystagmus. True nystagmus is rare: may be nystagmoid movements. (4) Optic neuritis, very rare and only slight.

BLOOD CHANGES.—Usually within normal limits. Polymorphonuclear leucocytosis recorded in 'myoclonic type' (Ellis).

CEREBROSPINAL FLUID.—Normal in at least one-third of cases. In about half, lymphocytosis is present: number of cells usually under 50 per c.mm., rarely as much as 100. Globulin may be slightly increased. Lange's colloidal-gold reaction may resemble tabes.

Clinical Types.—The 'positive' general and local nervous symptoms are usually associated, and commonly have an acute onset. So, also, the 'negative' symptoms are usually associated, and generally have an insidious onset. Combinations of symptoms, even with this limitation, are very numerous, and many clinical types have been described.

Diagnosis.—Often very difficult. Note mode of onset, fever, initial symptoms, and certain special symptoms, e.g., lethargy or excitement, ocular palsies, mask-like Parkinsonian facies (most constant symptoms). Diagnosis from and relations to:—

1. INFLUENZA.

i. Influenza and encephalitis lethargica have appeared at similar periods, e.g., 1890, 1918, and possibly 1837, but in 1916 the latter disease appeared before influenza commenced. The seasonal prevalence is different.

ii. Influenza is highly contagious, and encephalitis lethargica not obviously so.

iii. In encephalitis lethargica, pulmonary complications are very rare.

iv. In influenzal encephalitis, there is marked œdema of the brain, but (a) no perivascular infiltration, (b) only slight dilatation of vessels.

2. ACUTE ANTERIOR POLIOMYELITIS (Heine-Medin's disease). Main differences (Symonds):—

	Heine-Medin.	Encephalitis Lethargica.
Age	Mainly under 20 years.	All ages.
Onset	Acute or subacute.	Often insidious.
Temperature . .	Highest at onset before paralysis.	Often highest later (variable in character).
Distribution of lesions	Spinal cord, most.	Midbrain, most.
Involuntary movements	Absent.	Frequent, early or late.
Course .	Brief.	Prolonged.
Cerebrospinal fluid ..	Lymphocytes in early stages; globulin increased.	Lymphocytes often absent - rarely numerous; globulin slight.
Microscopic hæmorrhages	Constant and striking.	Inconstant and inconspicuous.
Perivascular infiltration	Slight.	Constant.

3. MENINGITIS, CEREBROSPINAL AND TUBERCULOUS.—By cerebrospinal fluid.

4. SYPHILIS.—By Wassermann reaction and symptoms.

5. CEREBRAL HÆMORRHAGE.—Gross hæmorrhages may occur in encephalitis lethargica and diagnosis be impossible.

6. BOTULISM.—Usually several cases in household. No pyrexia. *B. botulinus* may be present in stools.

Course and Prognosis.—

Mortality.—Excluding mild and abortive forms, about 33 per cent; including all forms is less than 20 per cent (exact figure doubtful). Deaths usually occur within first three weeks.

Duration.—Often many weeks or months. Mental impairment, paralyses, aphasia, and other changes have persisted in some cases so long as to be regarded as permanent, but recovery, when it occurs, is apparently usually complete (not yet certain).

Prognosis bad with: (1) Severe 'positive' symptoms, e.g., acute delirium; (2) High fever; (3) Pregnancy (to less degree).

Prognosis best with 'negative' symptoms, e.g., lethargy and diplopia.

Treatment.—Palliative and symptomatic. Hexamine usually administered. Lumbar puncture sometimes of apparent value. Netter advocates production of local abscess by injection of turpentine, 1 to 2 c.c., into thigh (value unconfirmed).

EPIDEMIC HICCOUGH.

Possibly a partial form of the 'myoclonic type' of encephalitis lethargica. Antispasmodics indicated, e.g., atropine, but treatment has little effect. Mortality low.

CHAPTER XXXVII.

HYDROPHOBIA.

(*Rabies. La Rage.*)

An acute, fatal, specific disease of the nervous system due to an unknown virus communicated to man through the saliva of an animal.

Distribution.—Widespread. Common in Russia and France. Not uncommon in United States. Rare in Germany. Was eradicated from Great Britain for many years as result of muzzling dogs. Australia free.

ANIMALS.—All mammals susceptible to inoculation, also birds. Dogs, wolves, and jackals most frequent naturally: cats, cattle, horses rarely. Propagation almost entirely by dogs in Russia also by wolves.

Morbid Anatomy.—*Nervous system* only affected. Other organs normal.

MACROSCOPIC.—Congestion and minute hæmorrhages may be present.

HISTOLOGY.—Several lesions occur.

(1) *Babes' 'rabic tubercles'*. Collections of round cells around the large cells of motor area in cord and bulb, chromatolysis and degeneration of the motor cells follow. Are not specific of rabies.

(2) *Van Gehuchten and Nélis*, 1900. In the peripheral ganglia of the central and sympathetic nervous systems, proliferation of endothelial cells occurs, destroying the nerve cells; final appearance not unlike sarcoma. *Method of examination*: remove plexiform ganglion of the vagus nerve; stain paraffin sections with hæmalum and eosin. Animal must be allowed to die of disease. *Value in diagnosis*. *Absence* of these changes *negatives rabies*: presence not quite conclusive, occurring rarely in old animals, but sufficient if symptoms suggestive.

(3) *'Negri bodies'*, 1903.—Bodies present within nerve cells, especially large cells of *cornu ammonis* (hippocampus major): shape and size variable, 1 to 25 μ. With Romanowski stains, are eosinophilic. With Giemsa, structure visible; within bodies are small granules.

Nature of Bodies.—In dispute. Theories: (a) Protozoa, and cause of disease (Negri). Probable. *Pro*: absent in health and other diseases, almost invariable in street rabies. *Con.*: absent in saliva, and in virulent brains of animals killed rapidly with 'virus fixé': never cultivated. (b) Cell degeneration (Babes).

Method of Examination (William and Lowden).—Make smear from brain tissue, fix, and stain with Giemsa.

Value in Diagnosis.—Are diagnostic.

The Virus.—Nature unknown. Possibly protozoon ('Negri bodies'). Present in all nerve tissues, and in saliva, reaching latter through nerves. Destroyed by drying in fourteen to fifteen days, and by light and heat. Passes a coarse Berkefeld or Chamberland filter. Spreads from site of inoculation entirely by nerves and nerve tracts. Absent in blood and solid organs.

Mode of Transmission.—In street rabies, by saliva from bites or licks: cannot pass through unbroken skin.

Symptoms.—

INCUBATION AND FREQUENCY OF DISEASE vary with—

AGE.—Shorter in children.

SITE OF INFECTION.—In order: (i) Face and head most severe, from richness of nerves and lacerated character of wounds; (ii) Hands, (iii) Other sites. Clothes protect considerably.

SEVERITY OF WOUNDS. Punctures and extensive lacerations most serious.

ANIMAL. In order: (i) Wolf—40 per cent bitten by wolves develop symptoms; (ii) Cat, (iii) Dog.

Frequency of disease after bites from rabic dogs: 16 per cent, if untreated.

INCUBATION PERIOD. Most often forty to fifty days. Earliest twelve days. Rare after three months. Over a year unproved. No symptoms. Wounds heal naturally.

Three subsequent stages are distinguished, often ill defined, and develop rapidly.

PREMONITORY STAGE.—Site of bite often becomes irritable, with pains in its neighbourhood. Depression, desire for solitude, intolerance of loud sounds and similar stimuli, with periods of irritability. Attacks of great *fear*. *Voice* becomes husky, and *difficulty in swallowing* commences. Temperature and pulse slightly raised. *Duration*, one to two days.

STAGE OF EXCITEMENT. Extreme irritability. Expression of terror.

SPASMS. Great severity and pain. Evoked by any stimulus. *Thorax and respiratory muscles* first affected. Laryngeal spasms especially caused by attempt to drink, by sight, or even mention, of *water*. Contractions of larynx may cause unusual noises. Often extreme dyspnœa. Later, spasms more general. *Saliva* abundant and viscid, cannot be swallowed and hangs from mouth. Between spasms, mentally clear. Maniacal attacks may occur, with attempts at biting.

Temperature, often up to 103°, rarely normal. *Pulse*, rapid. *Duration*, one-and-a half to three days.

STAGE OF PARALYSIS. Paralysis spreads and spasms cease. Unconsciousness, cardiac failure, death. *Duration*, a few hours.

TOTAL DURATION.—Usually four to five days.

CLINICAL TYPES.—(1) Furious rabies as above; (2) Paralytic or dumb rabies. Latter is very rare in man. Stage of paralysis alone present. May occur with extensive bites. Diagnosis only

Hydrophobia—Symptoms, *continued.*

possible by inoculation in animals: no Negri bodies. Possibly due to toxins. Common experimentally in animals.

Diagnosis.—

CLINICAL.—Usually simple in man. Diagnosis from :—

1. TETANUS.—Trismus, spasms not completely relaxed, nor specially evoked by water.
2. ACUTE BULBAR PARALYSIS.
3. LYSSOPHOBIA, PSEUDOHYDROPHOBIA.—In persons bitten by animals. Incubation period usually short. No temperature. Hysterical manifestations. Long duration.

PATHOLOGICAL.—(1) Negri bodies, *diagnostic*; (2) Van Gehuchten and Nélis' ganglionic changes, (3) Inoculation of brain or cord into animals.

EXAMINATION OF ANIMAL.—Clinical symptoms, autopsy (examination of cornu ammonis for Negri bodies), and inoculation of nervous tissue into other animals.

Prognosis.—Invariably fatal if symptoms develop. General mortality of cases bitten by dogs, 15 per cent (without inoculation). Efficient cauterization in 5 to 15 minutes halves mortality, of some, but slight, value after twenty-four hours.

RESULTS OF PASTEUR'S TREATMENT.—Mortality of all cases treated is less than 1 per cent. Early treatment important owing to occasional short incubation.

Prophylaxis.—Muzzling of dogs and destruction of stray dogs. No case occurred in Great Britain between 1903 and 1917.

Treatment.—

WOUNDS.—Early and thorough treatment most important. Make wound bleed, by opening thoroughly, bathe, *cauterize* with fuming nitric acid.

SYMPTOMATIC.—Palliative only. Avoid all stimuli. Opium and chloroform to ease spasms. Rectal injections.

Pasteur's Prophylactic Inoculation.—

BASIS.—

1. 'Street virus' has practically a constant strength. Inoculation kills a rabbit in fifteen to sixteen days.
2. Virus is exalted by passage through successive rabbits, until finally it kills in five to six days—'virus fixé'. (By passage through monkeys, virus *per contra* becomes attenuated.)
3. Rabbit's cord loses virulence on drying, being innocuous after fifteen to sixteen days. Hence a series of cords can be prepared of varying virulence according to period of drying.
4. Rabbits can be protected against an otherwise fatal injection by previous inoculation with such a series of cords, commencing with the weakest.

5. During the long 'incubation period' in man, similar inoculation can be performed.

METHOD.—Lengths of rabbits' cords, killed with 'virus fixé' and dried, are emulsified in normal saline and injected subcutaneously. First cord usually 8 days dried; length of 2 or 3 mm. used for injection. Intensity and duration of the course varies with nature of bites (fifteen to twenty-one days).

Symptoms during course.—May be pains near bites; rarely an ascending paralysis, very rarely fatal, possibly anaphylactic, or from attenuated virus.

DURATION OF IMMUNITY.—In dogs, diminishes about 20 per cent in a year. In man, no information: a second bite should be treated again.

Antirabic Serum.—Tizzoni and Centanni's, prepared by inoculating sheep with virus attenuated by peptic digestion (Italian method of immunization). The serum may be and often is employed in addition to the Pasteur treatment, especially in severe cases. Is protective to animals.

Rabies in Dogs.—Two types:—

FURIOUS TYPE ('*Street rabies*')—Stages practically correspond to human type. Early change in disposition, alternate excitement and desire for solitude, with increasing excitement. Voice alters, bark ending in *high plaintive note*, very suggestive. *Progress*: difficulty in swallowing food, not specially marked in regard to water. *Furious stage*: dog attacks everything, usually *runs straight*, may travel great distance. Paralysis and death follow. *Duration*: four to five days.

PARALYTIC OR DUMB RABIES.—Rarer. Early changes in disposition as above. No fury. Paralysis commences in jaw muscles, lower jaw falls. Hence unable to bite, and less dangerous. Paralysis extends. Death in two to three days. Not uncommon in rabbits killed with 'virus fixé'. Also occurs in dogs in Turkey.

CHAPTER XXXVIII.

RHEUMATIC FEVER.

An acute infection of unknown origin, characterized by multiple arthritis, and the frequent occurrence of inflammation of the endocardium of the valves of the heart, with resulting cardiac lesions.

There are certain important differences between rheumatic fever in adults and children. The general description applies to adults except where expressly noted, and the peculiarities in childhood are given in a separate section. The adult type commences about the age of 14 years.

Etiology.—Knowledge of etiological factors very incomplete.

CLIMATE. Universal, but especially in temperate climates. Prevalence highest in British Isles.

Rheumatic Fever—Etiology, *continued.*

SEASON.—In London, maximum in October and November, minimum in February and March. Varies greatly in different countries. In America, maximum in March. No relation to wet, dry, hot, or cold years can be traced statistically (Thompson and Greenwood).

AGE.—Most frequent from 15 to 35 years. Also occurs in childhood, but rare under 5 years and never under 2

SEX.—Males more common than females, except between 10 and 15 years.

HEREDITARY INFLUENCE. Generally accepted most marked in children.

PREDISPOSING CAUSES Enlarged tonsils and adenoids are probably important. Cold, wet, changes of temperature, fatigue may be factors even in good climates, but the rarity of rheumatic fever in France during the European war is noteworthy

IMMUNITY.—One attack definitely predisposes to others

MICROCOCCUS RHEUMATICUS Acute rheumatic fever has many resemblances to infections with micro organisms, but no causal bacterium has yet received general acceptance Triboulet and also Wassermann described a small coccus, the relations of which to rheumatic fever have been investigated, especially by Poynton and Paine It occurs commonly as a diplococcus or may be a short-chained streptococcus, and is Gram positive It has been isolated in rare instances from blood and joint fluids in rheumatic fever, and is observed in the valves in endocarditis. In animals, suppurative changes in the joints may follow inoculation.

Morbid Anatomy. No characteristic change in early cases *Joint changes* usually slight synovial membrane may show hyperæmia and swelling. No special changes in hyperpyrexia *Death* usually due to endocarditis or pericarditis

Symptoms. (Description applies to acute attack in an adult.)

PRELIMINARY SYMPTOMS Frequently none, but not uncommonly:—

SORE THROAT or acute tonsillitis. Often clears in a few days, and interval of perfect health up to two weeks may elapse

IRREGULAR JOINT PAINS with slight malaise for a few days

ONSET.—Abrupt Chill but no rigor Condition fully developed in twenty-four hours

CHARACTERISTIC SYMPTOMS are (1) *Joints* swollen and painful, (2) *Face flushed*, (3) *Profuse sweats* with very sour odour, even in absence of sweats the skin is moist, and in spite of pyrexia is never dry, (4) *Sore throat*, (5) *Temperature high* 101° to 103°, (6) *Pulse* soft and rapid, 100 to 120. Ordinary febrile symptoms marked Tongue furred and moist Anorexia, constipation, febrile urine, thirst Sudamina and miliaria frequent. Mind clear. Pain may cause sleeplessness

JOINT AFFECTION (*Arthritis*).—Characteristics are:—

MULTIPLE JOINTS affected; especially larger joints, often

symmetrically. In severe attacks, many attacked simultaneously.

FREQUENCY OF INVOLVEMENT.—Order: (1) Knee, (2) ankle, (3) wrist, (4) elbow, (5) shoulder; vertebral, sternoclavicular, jaw, and phalangeal joints very rare.

INFLAMMATION.—Wanders from joint to joint, e.g., as knee recovers, wrists swell. Change may occur in twenty-four hours. In space of three or four days many joints may have been affected.

JOINTS.—Swollen, red, hot to the hand, tender, and extremely painful on movement. Changes are mainly inflammation of peri-articular tissues. The synovial membrane often palpable. Tissues are infiltrated with serum, but œdema and pitting of the skin on pressure is absent even in severe cases. Tendon sheaths involved. *Extensive effusion into joint rare.*

JOINT FLUID.—Turbid. Contains numerous polynuclear leucocytes, but never has appearance of pus. *Suppuration never occurs.*

Directly acute symptoms subside, joint usually appears normal.

TEMPERATURE.—Rise rapid, 101° to 103° or 104°; rarely higher. Irregular. Falls gradually. First recorded temperature usually the highest, owing to subsequent administration of salicylates. Persistence of pyrexia after five days' treatment with salicylates suggests endocarditis or pericarditis.

HEART. Systolic murmur frequent at apex. May be: (a) *Myocardial*, disappearing on treatment; (b) *Endocardial*, subsequently progressing and becoming permanent.

PULSE.—At onset 100 to 120. Soft. Tracings often show slight irregularity. Falls with temperature. With salicylates may become very slow, 40 to 50, but this is of no importance.

URINE.—Febrile type. Trace of albumin occasionally.

BLOOD. Polynuclear leucocytosis. Secondary anæmia develops rapidly.

Progress. In absence of complications and without drugs fever and acute symptoms subside in about ten days. With salicylates rarely exceed four days.

Subacute Rheumatic Fever. No definite dividing line from acute. All symptoms less marked. Duration may be long. Cardiac lesions common.

Relapses. Very frequent: in 15 per cent of first or second attacks.

Complications. Those of importance are (1) Hyperpyrexia; (2) Cardiac; (3) Pulmonary; (4) Nervous; (5) Cutaneous; (6) Rheumatic nodules.

HYPERPYREXIA. Extremely rare. Never in children under 12. Usually in second week of first attack. Temperature may rise to 108°. *Delirium* or *pericarditis* commonly present (*see* NERVOUS COMPLICATIONS). Pulse feeble, stupor and death.

CARDIAC LESIONS. Though described here as a complication, cardiac changes are truly a part of the disease as much as arthritis.

Rheumatic Fever—Complications, *continued.*

ENDOCARDITIS.—Most serious feature of rheumatism. For symptoms, etc., *see* ENDOCARDITIS. Special features:—

Frequency about 50 per cent of cases. Increases with number of attacks, diminishes with age. Children rarely escape.

Valves commonly affected, in order of frequency: (1) Mitral alone; (2) Mitral and aortic; (3) Aortic alone—rare.

Mitral stenosis only develops slowly, and hence is not recognizable during acute stages of a first attack

Pathological changes are of simple endocarditis: verrucose and infective form rare during attack of rheumatism.

Subsequent progress.—Signs and symptoms of endocarditis slight in first attack, but pathological changes tend to advance after attack of rheumatism has passed.

Mortality low during the acute attack.

PERICARDITIS.—Frequently associated with acute rheumatism, especially in children (*see* PERICARDITIS). Special features: (1) Occurs in about 10 per cent of cases, sexes equal; (2) Slightly more frequent in first attacks, but mortality in first attack is 40 per cent and in second 10 per cent, (3) May occur at any time during attack, with or without endocarditis; (4) Effusion may occur (20 per cent of cases), *but is never purulent*. (5) Delirium occasionally, and hyperpyrexia rarely, (6) Arthritis usually severe.

In fatal cases, endocarditis also nearly always present.

MYOCARDITIS.—Probably frequent, leading to dilatation. No definite signs.

PULMONARY COMPLICATIONS.—Pneumonia and pleurisy are common with pericarditis; rare in other forms. Dry pleurisy occasionally occurs, but effusion very rarely.

NERVOUS COMPLICATIONS.—Apart from chorea, are extremely rare.

DELIRIUM ('Cerebral rheumatism').—Occurs with hyperpyrexia or pericarditis (or both together). Probably never independently. Delirium may be active or quiet. Passes into coma. Mortality very high.

CHOREA.—Associated with rheumatism not uncommonly, but mainly in children.

MENINGITIS.—Very rare.

CUTANEOUS COMPLICATIONS.—In acute stage skin is moist. Drenching sour sweats were characteristic before introduction of salicylates.

ERYTHEMATA.—Frequent in children.

PURPURA.—May occur at onset, especially in children (Joint pains are not infrequent with purpura of any type, and association is not definite proof of acute rheumatism)

ERYTHEMA NODOSUM.—From frequency of various erythemata in rheumatic fever erythema nodosum is widely accepted as rheumatic, but connection is not proved. Endocarditis never follows, and definite arthritis is rare.

RHEUMATIC NODULES.—Occur on fibrous tissue and periosteum of bones lying close under the skin, e.g., olecranon, tendons and fasciæ especially about elbows and wrists, also on scapulæ and vertebræ. Number usually three or four, rarely twenty to thirty, occasionally very numerous. Best recognized by drawing skin tight and palpating gently. Almost confined to children Pericarditis has been observed subsequently in many cases.

Diagnosis.—Usually simple. Diagnosis in adults from:—

ACUTE ARTHRITIS DEFORMANS (osteo-arthritis).—Tends to attack smaller joints. Chronic articular changes

SECONDARY ARTHRITIS.—Septic arthritis in pyæmia and septicæmia Gonorrhœal arthritis (*q.v.*). Rare. scarlet fever, dysentery.

GOUT.—Age of patient; etiology, previous attacks; small joints usually affected, especially great toe and thumb.

Mortality. In acute attacks, very low, not exceeding 2 to 3 per cent, which is mainly due to cardiac complications Great indirect mortality from cardiac lesions Hyperpyrexia has high mortality, but is very rare

Treatment. Indication is to protect heart specially

REST IN BED At least four weeks after temperature normal, between blankets in early stages Initial purge

DIET Milk and milk foods for three weeks Fluids and lemonade in large amount

LOCAL TREATMENT Wrap limbs in warm cotton wool Cradle to support weight of bed clothes If much pain, paint with methyl salicylate, or use sodium bicarbonate fomentations

SALICYLATES Sodium salicylate recommended for routine use to be prescribed with sodium bicarbonate

℞ Sodii Salicyl gr xx Fract. Zingiberis ♏ xx
Sodii Bicarb gr xl Aq. Chloroform. ad ℥j

Two hourly for 6 doses. Four hourly until temperature is Three times daily for further three weeks

Aspirin or salicin may be used if temperature does not fall, especially in children

Action of Salicylates Usually rapid Ease the articular pains, and cause fall of temperature It is doubtful whether they lower the incidence of endocarditis

PAIN SEVERE Nepenthe or Dover's powder.

HYPERPYREXIA Hydrotherapy, as in typhoid fever.

ENDOCARDITIS See ENDOCARDITIS

Rheumatic Fever in Children.—Differs from clinical condition in adults by more insidious character Does not occur under 2 years of age

ARTICULAR LESIONS Often slight and overlooked and endocarditis often progresses to mitral stenosis and incompetence without any illness being observed. Recurrent tonsilitis or sore

Rheumatic Fever in Children, *continued.*

throat may be the only manifestation, or possibly endocarditis may occur without other symptoms.

COMPLICATIONS are more common in childhood: chorea, pericarditis, rapid anæmia, and also subcutaneous nodules.

DIAGNOSIS.—From :—

ACUTE OSTEOMYELITIS.—Constitutional symptoms very severe. Pain is not in joint.

ACUTE POLIOMYELITIS. May be associated with hyperæsthesia.

INFANTILE SCURVY. - Age under 2 years.

CONGENITAL SYPHILIS. -Occurs as (1) *Syphilitic epiphysitis*: age under 2 years, affects epiphyses and not joints (2) *Symmetrical synovitis*, painless, at age of puberty.

STILL'S DISEASE. – Rare. Juvenile form of arthritis deformans. Chronic. Spleen and glands often enlarged. Heart unaffected.

CHAPTER XXXIX.

ACUTE CORYZA.

(Acute Rhinitis. Common 'Cold.')

Acute inflammation of mucous membrane of upper air-passages.

Etiology.—

DISTRIBUTION. Widespread in temperate and cold regions

SEASONS.—Especially at changes of temperature, as in early winter and early spring.

AGE - No age immune: children very susceptible.

BACTERIOLOGY. Various organisms may predominate, e.g., *Micrococcus catarrhalis, Streptococcus, Staphylococcus, Pneumococcus.*

INFECTIVITY Varies greatly with (1) Individual: marked idiosyncrasies. (2) Outbreak: schools and households often affected

Symptoms.—

INITIAL STAGE. Chill, sneezing, head feels heavy, skin dry.

NASAL MUCOUS MEMBRANE.—

First Stage, congested; unable to breathe through nose; duration one to three days. *Second Stage*: watery discharge, duration two to seven days. *Third Stage*: mucopurulent discharge; gradually subsides.

Inflammation often spreads to: (a) *Tonsils*. Sore throat, common initial symptom (b) *Pharynx*: Swallowing painful. (c) *Larynx*. Voice husky. (d) *Eustachian tubes*: Deafness (e) *Conjunctiva and tear ducts*: Eyes 'run'. (f) *Œsophagus.*

Temperature and pulse moderately raised.

Smell, taste, and appetite affected. Constipation common.

Extension to trachea or bronchi produces 'cold on the chest' (acute bronchitis).

Treatment.

INITIAL STAGE. -No treatment is reliable to abort attack

Drugs: quinine (tinct. quin. ammon. ʒj. t.d.s., for adult), Dover's powder gr. x, at night; aspirin; cinnamon, camphor.

Following is often efficient, if taken early in attack:—

℞ Vin. Ipecac. ♏v to ♏x | Liq. Ammon. Acet. ʒij to ʒiv
Tinct. Camph. Co. ♏xx to ♏xxx | Aq. Chloroformi ad ℥j
Spt. Ætheris Nitrosi ʒss to ʒj |

Every 4 hours.

CONGESTED AND DISCHARGING STAGES. Methods to encourage perspiration: hot drinks with drugs as above; hot bath (with caution). If bronchitis, rub with camphorated oil (*see* ACUTE BRONCHITIS).

For *mucopurulent discharge*: Alkaline nasal douche, snuffed into nostrils from the hand, or from atomizer:

℞ Sod. Bicarb. gr. xx | Glycerini ʒj
Boracis gr. xx | Aq. ad ʒiv

Mix with equal amount of warm water.

Bed for two days in earlier stages if severe. Brisk *purgative*. Light diet.

In convalescence iron and strychnine tonic, cod liver oil.

VACCINE TREATMENT. For recurrent colds. Of prophylactic value only, but effective in some cases. Prepared from culture from patient's mucous membrane. Stock vaccines are also in use, containing streptococcus, pneumococcus, and influenza bacillus.

PROPHYLAXIS. For recurrent colds examine nose and throat for enlarged tonsils, etc. Protect against chills, in moderation.

Diagnosis.—Measles commences with typical coryza.

CHAPTER XI

GLANDULAR FEVER.*

An acute infectious disease of children characterized by marked enlargement of cervical lymphatic glands. Described by Pfeiffer in 1889. Probably by no means uncommon and often overlooked or diagnosed erroneously.

Etiology.—Virus unknown. Mainly affects children between 5 and 9 years. Adults not immune, but symptoms less typical; usually parents of affected children. Infectious, and may occur in small epidemics, usually in spring. Is a disease *sui generis*. Is not anomalous mumps, as many have this previously or subsequently, and salivary glands are never affected, nor an anomalous exanthem, as no rashes occur even in epidemics.

Symptoms.—

ONSET.—Sudden. Temperature 101° to 103°. Usual febrile symptoms of childhood. Constipation often obstinate.

ON SECOND OR THIRD DAY.—*Lymphatic glands rapidly*

* *See Brit. Med. Jour.*, March, 1921.

Glandular Fever—Symptoms, *continued.*

enlarge behind sternomastoid, about the middle of its length: form a large mass: on palpation several discrete glands: not always painful. Other glands enlarging may be posterior cervical; less often axillary and inguinal. Mesenteric glands not uncommon, and may be severe abdominal pain. Spleen often palpable also liver. Salivary glands never involved, but superficial submaxillary and pre-auricular lymphatic glands may be enlarged. Fauces, slight reddening: no tonsillitis: may be some pain on swallowing, but changes slight compared with glandular enlargement. No rash.

PROGRESS.—Often after three or four days glands subside rapidly May be unilateral at onset, and other side enlarge a few days later with recurrence of symptoms.

BLOOD.—May be moderate leucocytosis, with a high percentage of lymphocytes (up to 80 per cent). not always present

DURATION.—Two or three weeks Convalescence slow owing to weakness, often a resulting anæmia, and some degree of debility usually persists for many months Mortality negligible.

Complications.—*Hæmorrhagic nephritis* not infrequent apparently only transient. Suppuration of glands very rare Cervical glands may remain palpable for many months.

Diagnosis.—Often mistaken for tuberculous glands note rapid subsidence. Also from acute leukæmia, mumps, Hodgkin's disease, syphilis, and secondary infections from the fauces

Treatment.—Symptomatic: bowels should be moved Tonics and avoidance of strain during convalescence.

CHAPTER XLI.

TRENCH FEVER.

An infectious disease due to an unknown virus transmitted by the excreta of lice, and characterized by an initial febrile period, a tendency to relapses and periodic pyrexia, and frequently by hyperæsthesia of the shins. Never fatal

History.—Occurred during the war to an enormous extent among troops on active service. Few cases among civilians and disease has now disappeared.

GRAHAM, 1915, first published accounts of a pyrexia agreeing with no known disease.

HUNT and RANKIN, 1916, described further cases

McNEE, RENSHAW, and BRUNT, 1916, described the condition more fully, and its transmission by the blood of infected patients.

Mode of Transmission.—The transmission by lice became suspected. The following facts are established, but virus has not been discovered:—

1. Virus is present in blood of patients: can be transmitted by inoculation of blood or of plasma, hence is not intra-

corpuscular. Is present also in urine and sometimes in saliva, but not in fæces. Virus can pass a Chamberland L filter. Killed in an hour at 70° C. but not at 60° C.

2. Transmission by lice fed on trench-fever patients: (a) Bites do not convey infection; (b) Excreta of lice inoculated by scarification convey the infection, (c) Lice crushed and rubbed into scarification convey the infection.
3. Lice after feeding are not infectious for five days, i.e., a cycle occurs in body.
4. Lice are infective for at least twenty-three days.
5. Virus is not transmitted to offspring.
6. Virus is not normally present in lice.
7. Virus has been transmitted from a patient eleven weeks after attack.

Incubation period.—About two to three weeks under ordinary conditions. By scarification: about eight days. By simple transference of lice to healthy persons: fourteen to thirty-eight days.

Types of Fever.—Several types of trench fever were described originally, as a short form, a long form, and a relapsing pyrexia. It is now probable that the relapsing pyrexia is invariably preceded by an initial febrile attack. The long and short forms practically vary only in duration, and may be grouped as the initial fever.

Symptoms of Initial Fever.—

ONSET.—Sudden. Often previous malaise is present for two or three days.

GENERAL SYMPTOMS. (1) *Headache* severe, frontal and at back of eyes. (2) *Giddiness.* (3) *Pains in back and legs.* (4) *Sweats*, often profuse. (5) *Face* flushed. (6) *Conjunctivitis* common. (7) General febrile symptoms: anorexia, constipation, shivering, but no definite rigors, vomiting occasionally. (8) Herpes labialis occurs occasionally. (9) *Spleen* enlarged in about one-third of the cases. Usually tender. (10) *Tenderness and pains in shins*: most characteristic symptom, often very acute. Usually not present in first few days. Especially lower half of shins. Pain, also severe, may occur in thighs and knees; sometimes in calves, but this often entirely absent. (11) *Rash*: pale pink irregular erythematous or roseolar spots, do not project, disappear readily on pressure. Do not occur in more than one-third of the cases, and usually not until relapses or the periodic rises. Formerly mistaken for enteric spots. (12) *Blood*: may be moderate leucocytosis.

Pyrexia.—

A. INITIAL FEVER.—

SHORT FORM.—Duration usually three to six days; often fluctuates; then falls to normal. *Relapses* very frequent. *Temperature* may be irregular, subsequently, sometimes for long periods, even in absence of definite relapses.

LONG FORM.—Duration six to twenty days.

Trench Fever—Pyrexia, *continued.*

B. RELAPSING PYREXIA.—

Periodic rises occur, usually at five-day intervals: feels well in intervals

Febrile period one to two days: temperature 102° to 104°, pulse rapid. Symptoms as in initial fever.

Occurs in small proportion of cases. May follow directly on initial fever or after interval of weeks. Initial fever may be overlooked.

Attacks tend to diminish; duration of pyrexia may be very short, a few hours only, may be unnoticed (or possibly absent), while malaise, increased pulse, and other symptoms occur.

Identical febrile attacks may occur many months after infection.

Sequelæ.—(1) Slight febrile attacks. (2) Myalgia; (3) Tachycardia. (4) Debility may result from the constant shin pains and pyrexia. Endocarditis never results.

Progress.—Never fatal. Shin pains often persist after other symptoms have subsided. Complete recovery finally.

Treatment. Confine to bed at least three weeks in initial attack; main object is to save the heart. No drug has any effect on temperature, or on the shin pains if severe.

CHAPTER XLII

SANDFLY FEVER.

(Phlebotomus Fever. Pappataci Fever. Three day Fever.)

An acute fever of three days' duration caused by an ultramicroscopic organism introduced by the bite of the sandfly or phlebotomus.

Geographical Distribution.—Eastern Mediterranean coast.

Mode of Infection.—DOBELL, 1908, proved that infection followed the bites of sandflies, and reproduced the disease by injecting his blood into a healthy subject.

Symptoms.—*Incubation*: one to six days. *Invasion*: Rigor, severe headache, malaise, general pains. Temperature 104° to 104°, rising in twenty-four to thirty-six hours. Then defervescence, may be accompanied by sweating, vomiting, or diarrhœa. *Duration*: three days. No eruption. No desquamation. No recrudescence. No complications. No sequelæ, except some weakness. Never fatal.

Prophylaxis.—General sanitary measures diminish prevalence of sandflies. Small size of fly enables it to pass through mosquito netting.

Section I.- **Specific Infectious Diseases,** *continued.*

F. DISEASES DUE TO SPIROCHÆTES.

CHAPTER XLIII.

RELAPSING FEVER. AFRICAN TICK FEVER.

RELAPSING FEVER.

An acute febrile disease caused by a spirochæte and characterized by alternate periods of fever and apyrexia of five to ten days duration.

Distribution. Occurs in all continents, with slight differences in spirochætes, mode of transmission, and symptoms. In Europe known as 'famine fever' or 'seven day fever', lingers in Ireland. Widespread in India. Not of recent years in United States. In Africa known as 'tick fever'.

Spirochæte. *Sp. obermeieri* discovered in blood by Obermeier in 1873; invariably present during febrile periods, but not in intervals. It is unknown whether periods correspond to developmental phases (as in malaria). Length 15 to 40 μ; numerous spirals; actively motile, by lashing movements or action of spirals.

Mode of Infection. *By lice.* Infection experimentally, never follows bites of lice, but occurs if bodies are broken and rubbed into scratches; probably usual method. Eggs of infected lice can transmit infection through several generations. Animals are susceptible experimentally but not naturally infected. Laboratory infection occurs very readily from touching infected material or blood.

Morbid Anatomy. No special changes except enlargement of spleen and liver.

Symptoms.—

INCUBATION. Usually five to seven days. Slight malaise two to three days before invasion.

INVASION.—*Sudden onset*: rigors, headache, sweats, intense pains in long bones, giddiness, and often vomiting. *Temperature* 103° to 104° on first day. Pulse 110 to 180. Spleen enlarges, also liver. Slight jaundice, constipation or diarrhœa. Occasionally herpes. *Blood*: spirochætes present, polynuclear leucocytosis.

CRISIS.—Usually fifth to seventh day of fever. Sweating, rapid apyrexia. Death at crisis may occur in weakly persons.

APYREXIAL PERIOD. Duration about same as fever; rapid improvement, followed by—

RECURRENCE.—About fourteenth day. Similar to initial attack, but usually milder. Rarely more than one recurrence in European type; occasionally three or four. Absence of recurrence rare.

Relapsing Fever—Symptoms, *continued.*

CONVALESCENCE.—Slow, owing to exhaustion.

Complications.—Not common. Delirium during fever. During convalescence: rarely iritis, meningitis, paralyses, convulsions.

Prognosis.—In good conditions, mortality under 2 per cent, especially with modern treatment. With overcrowding and bad hygiene, rises to 20 or 30 per cent. One attack does not protect.

Diagnosis.—During febrile period, spirochætes in blood. In a febrile interval, blood will agglutinate spirochætes in infected blood (equal drops: incubate at 37° C. for half hour). In doubtful cases injection of blood into rats or monkeys (25 c.c.).

When treated with quinine in absence of blood examination, crisis may lead to diagnosis of malaria.

Treatment.—Arsenobenzol preparations are specific. Spirochætes disappear, and temperature falls in few hours. Recurrence occasionally happens: inject smaller amount. In absence of these drugs, general treatment of fevers, cold sponging, etc. Pain often needs morphia. At crisis, stimulants necessary, especially in old or weakly persons.

PROPHYLAXIS.—Factors promoting spread are similar to typhus: ove crowding and lice. Sterilization of clothes, cleanliness of dwellings, protection from lice.

AFRICAN TICK FEVER.

An acute febrile disease caused by a spirochæte and characterized by alternate periods of fever and apyrexia of two to three days' duration. Closely allied to relapsing fever (*see* p. 257). The following special features may be noted:—

Spirochæte.—*Sp. duttoni*: first studied fully by Dutton and Todd in East Africa, and by Koch, though previously observed by others. Differs very slightly from *Sp. obermeieri* of relapsing fever, but more pathogenic to monkeys and other animals; also slight differences when cultivated by Noguchi's method. Immunity to *Sp. duttoni* does not cause immunity to *Sp. obermeieri*, or vice versa.

Mode of Transmission.—*By a tick, Ornithodorus moubata*, probably not by salivary glands, but by secretion of special coxal glands when feeding. In the body of the tick the spirochæte undergoes morphological changes, forming minute chromatin granules, which are a phase in the life history and convey the infection (Leishman). Eggs of infected ticks can transmit infection through several generations.

Clinical Course.—Resembles relapsing fever, but pyrexial periods are shorter, two to three days, may be numerous relapses, and spirochætes are more scanty in the blood.

Mortality.—Low; but arsenobenzol preparations less effective in this type.

CHAPTER XLIV.

EPIDEMIC JAUNDICE.*

(*Spirochætosis Icterohæmorrhagica. Weil's Disease.*)

An acute condition due to a spirochætal infection, occurring in local epidemics, and characterized by fever, jaundice, enlargement of the liver, hæmorrhages, and frequently a secondary fever.

History.—Epidemics of jaundice long recognized. Described by Matthieu and later by Weil in 1886. Spirochæte discovered by Inada and others in Japan in 1914. Many cases in France during the war. Probably several types: some due to a spirochæte non-pathogenic to animals.

The Spirochæte. Length 5 to 25 μ. In stained preparations, 4 to 5 waves; with dark-ground illumination, numerous fine spirals; by special methods, characteristic flagellum with terminal 'knob'.

CULTIVATION.—Feasible in many media, e.g., diluted rabbit's serum with covering of liquid paraffin. Subculture in two to three weeks.

DISTRIBUTION IN HUMAN BODY. In peripheral blood up to fifth, and rarely ninth, day of disease. Later, excreted in urine. Occurs in liver, suprarenals, and, later, kidneys, but scanty in all human organs. Absent in life from duodenal contents.

MODE OF TRANSMISSION.—Epidemics usually local, in wet mines and wet trenches. *Rats* are often infected without symptoms of disease: spirochætes mainly in kidney. Human and rats' urine may be mode of propagation.

Morbid Anatomy.—

LIVER. Enlarged: ordinary changes of catarrhal jaundice: less often necrosis and degeneration, which may be extreme, as in cholæmia and acute yellow atrophy.

DUODENUM AND BILE PASSAGES.—May be slight inflammation, but no proof of obstruction.

LUNGS.—Hæmorrhages, often of considerable size.

SPLEEN.—Enlarged.

KIDNEY.—Often parenchymatous nephritis.

BLOOD.—Fragility of red cells not increased.

Symptoms.—

ONSET.—Sudden. Shivering, headache, *marked prostration*, and muscular pains.

EARLY SYMPTOMS.—Temperature 103° to 105°. Pulse rarely exceeds 100. Anorexia. Constipation, rarely diarrhœa. Vomiting. Tongue furred.

* Clinical description by Dawson, Hume, and Bedson, *Brit. Med. Jour.*, Sept., 1917.

Epidemic Jaundice—Symptoms, *continued.*

JAUNDICE.—Begins on fourth or fifth day, maximum about ninth day.

HÆMORRHAGES Rarely absent in severe cases may be from lungs, stomach, nose, rectum, or as purpura.

HERPES LABIALIS —Frequent.

LIVER --Enlarged and tender Spleen rarely palpable

BLOOD.—Total leucocytes 20,000 to 30,000 Polynuclear cells 80 to 90 per cent

URINE — Bile present for three to four weeks Albumin and casts common Acetone only with cholæmia

PROGRESS *Initial fever* falls in ten to fourteen days Symptoms improve *Secondary fever* common in third week may be 103°, about ten days no return of symptoms

SUBSEQUENT COURSE Usually uninterrupted Convalescent in three to five weeks.

ANOMALOUS FORM Apparently occurs with similar symptoms, but *jaundice absent*

Diagnosis.—

CLINICAL Suggested by sudden onset, pyrexia, prostration herpes, and jaundice about fourth day In enteric, jaundice rare especially before second week From catarrhal jaundice by late onset of jaundice, but often clinically impossible

PATHOLOGICAL By intraperitoneal injection of guinea pigs

1 BLOOD Spirochætes present until fifth day and rarely to ninth Direct observation difficult (Burri's Indian ink or Fontana's silver method) *Guinea pig* Intraperitoneal injection of 3 to 5 c c of patient's blood incubation period six to thirteen days, then jaundice collapse and death in twenty four hours, may be petechial hæmorrhages spirochætes present in blood and solid organs especially liver, also kidneys and suprarenals Hæmorrhages in lungs and intestinal walls spleen enlarged acute parenchymatous nephritis

2 URINE —Spirochætes present, not before tenth day almost invariably present by twentieth day rare after fortieth day Centrifugalize urine and examine deposit

Mortality. Very low Death occurs with convulsions and signs of cholæmia

Treatment.- No specific treatment Salvarsan preparations of no effect Aperients necessary

Epidemic Catarrhal Jaundice.--Jaundice occurred epidemically during the war on the Eastern fronts Spirochætes not present, also slight clinical differences from spirochætosis icterohæmorrhagica.

CHAPTER XLV.

SYPHILIS:

A specific infection by the *Spirochæta pallida*,* acquired by contact, commonly sexual intercourse, or transmitted through the mother. The essential lesion is an infective granuloma.

Introduction into Old World from America in 1493 is generally accepted.

Name 'syphilis' appears first in 1530, in a poem by Fracastor. 'Syphilus' was the name of the infected hero.

SCHAUDINN, 1905, discovered the *Spirochæta pallida*.

WASSERMANN, 1908, described the original serum test based on the Bordet-Gengou reaction.

EHRLICH, 1910, produced salvarsan as a cure.

The Parasite.—

MORPHOLOGY. A very delicate organism, often somewhat curved; length 4 to 14 μ, breadth 0·25 μ; numerous fine sharp regular cork-screw spirals, commonly eight to twelve in number, persisting during rest and after staining. Flagella stained by special methods, one at each end. Motile but not very active, movements being: (1) Rotary about long axis; (2) Backward and forward movements; (3) Bending movements. Change in position slight. Does not pass a Berkefeld filter.

LIFE HISTORY. Unknown.

OCCURRENCE IN THE BODY. Spirochetes are extracellular.

PRIMARY LESIONS. Presence most numerous in primary sore, condylomata, mucous patches.

SECONDARY LESIONS. In cutaneous eruptions, scanty.

GUMMATA. Scanty, rarely found.

NERVOUS SYSTEM.—In tabes and general paralysis, recently demonstrated by Noguchi, very scanty.

CONGENITAL INFECTIONS. Often extremely numerous in tissues, especially in liver.

Have been found in placenta, umbilical cord, and with difficulty in blood of infected persons.

CULTIVATION. The parasite has been cultivated by Noguchi, strictly anaerobically, in ascitic fluid and agar containing a piece of sterile rabbit kidney or testis.

TRANSMISSION TO ANIMALS. *In higher apes*, by scarification and inoculation; subcutaneous inoculation negative. Primary lesion after thirty days, resembles human lesion with induration of glands. Secondary lesions mild; occur in about 50 per cent. No tertiary lesions. Wassermann reaction positive. In *lower*

* Correct designation is *Treponema pallidum* or *Spirosoma pallidum*. Schaudinn's original name *Spirochæta* (or *Spirochete*) *pallida* is still most commonly employed.

Syphilis—The Parasite, *continued.*

monkeys and rabbits: local sore only In rabbit's eye, produces iritis and keratitis

METHODS OF OBTAINING SPIROCHÆTES —

Chancre.—Wash with normal saline: if painful, swab with 4 per cent eucaine: suck with small Baer's flask, or squeeze with *protected fingers* to obtain deep fluid: transfer fluid to slide with platinum loop

Glands —Puncture groin glands with hypodermic needle

METHODS OF EXAMINATION —(Oil immersion lens for all methods)

1. BURRI'S INDIAN INK METHOD —Fluid from lesion stirred in drop of Indian ink ('Chin chin liquid pearl'), and spread on slide Examined with artificial light Spirochæte appears as white shining spiral on black ground
2. STAINED FILMS —Fluid spread on slide Best stained with Giemsa's ~~aqua-eosin~~ *Sp pallida* stains a pinkish violet does not stain with ordinary dyes
3. DARK GROUND ILLUMINATION —Special paraboloid condenser Morphology and motility of spirochæte well exhibited

SECTIONS. Levaditi's silver deposition method Tissue impregnated with silver nitrate then reduction by pyrogallic acid deposits silver on the spirochætes sections cut by microtome

IDENTIFICATION OF SP PALLIDA —Mainly by —

1. Number (8–12) and regularity of spirals Parasite very fine
2. With Giemsa, stains faint pinkish violet Other spirochætes stain deeper blue

Sp refringens, present in ulcerated lesions (*a*) Thicker and coarser, (*b*) Few, irregular, and flatter spirals, (*c*) Stains deeper and more blue, (*d*) Greater motility

Morbid Anatomy.—All syphilitic lesions have in common (1) Inflammation of connective tissue, (2) Changes in vessels endarteritis or periarteritis The picture varies with site of the lesion

PRIMARY CHANCRE.—Consists of (1) Connective tissue cells and fibroblasts, (2) Infiltration of small round cells, (3) Epithelioid cells and giant cells (scanty) Small vessels show obliterative endarteritis causing the surrounding induration

GUMMA —An 'infective granuloma'. Consists in early stage, of cells as above *Early* vessels scanty *Later* new vessels numerous. Then obliterative endarteritis occurs, followed by caseation of tissue, necrosis, and rupture in certain situations.

Distinction from tubercle difficult Main points (1) In gumma, new vessels prominent, in tubercle absent, (2) In gumma, epithelioid and giant cells scanty, and latter less definite than in tubercle.

Modes of Infection.—

SEXUAL CONNECTION. —Common sites of chancre *Male*, sides of frenum, glans, sulcus, prepuce; less commonly, within meatus, body of penis, scrotum, etc. *Female*, labia minora, os uteri; occasionally labia majora; vagina rare.

ACCIDENTAL INFECTIONS.—*Extragenital chancres.*
In medical practice, e.g., on fingers or back of hands.
Lips: commonest extragenital chancre: kissing, or from infected articles.
Occasional sites: Nipple in wet-nurses, tonsils in glass blowers.
Various sites by accidental infection or in sexual perversion.

CONGENITAL INFECTION.—Intra-uterine infection of fœtus through placenta. Mother often has no signs, but Wassermann reaction is positive.

Two NOTEWORTHY LAWS.—(1) *Colles's Law*: A syphilitic infant does not infect its own mother. (2) *Profeta's Law*: A mother with syphilitic symptoms may suckle her own infant without infecting it.

Syphilis thus may be ACQUIRED or CONGENITAL.

ACQUIRED SYPHILIS.

Incubation Period.—Interval between infection and appearance of primary lesion (chancre) usually two to three weeks. Rarely under ten days. To be doubted if under one week or over six weeks. Period often impossible to determine.

Stages of Syphilis.—Symptoms are referred to three stages, *Primary, Secondary, and Tertiary.* Certain late nervous lesions are known as quaternary syphilis or parasyphilis.

Primary Stage.

The Chancre.—
Initial lesion of syphilis is the 'primary', 'hard', or 'Hunterian' *chancre*, a local manifestation which commences as a painless small red papule: enlarges to size of pea: ruptures, forming small ulcer.

CHARACTERISTICS. Raised, edges indurated, may feel like nodule of cartilage, floor often greyish slough, secretion slight, suppuration uncommon. Freely movable.

PROGRESS.—Granulation occurs, and ulcer heals with or without treatment.

SCAR.—May be slight or absent.

USUALLY SINGLE, occasionally two or, rarely, more.

SITE.—*See* MODES OF INFECTION, p. 262.

Varieties of Chancre.—On glans induration often absent. A tight prepuce becomes œdematous, chancre palpable below. In females, often obscured by œdema: frequently unnoticed.

SEPSIS.—With infection by septic organisms or bacillus of soft sore, acute ulceration occurs, very painful: diagnosis obscured.

EXTRAGENITAL CHANCRES.—Induration less marked: ulceration greater.

PHAGEDÆNA.—Rare: rapid ulceration, penis may be destroyed.

Lymphatic Glands in area of chancre (e.g., groin) enlarged and 'shotty': suppuration only with septic infection.

Diagnosis.—Especially from: (1) Traumatic ulceration; (2) Soft sore; (3) Herpes of prepuce; (4) Scabies.

Syphilis—Acquired, *continued.*

Secondary Stage.

Is a period of general infection, a long-drawn fever with constitutional symptoms, as opposed to the localized lesion of the primary stage.

Onset.—Within four to twelve weeks of coitus. Usually five to six weeks after chancre. (Scientifically, commences with the positive Wassermann reaction.)

Duration.—About two years, but no definite limit.

Principal Manifestations.—(1) Rash; (2) Sore throat; (3) Mucous patches; (4) Condylomata; (5) General enlargement of lymphatic glands; (6) Loss of hair; (7) Anæmia; (8) Fever; (9) Headache and insomnia not uncommon.

OTHER MANIFESTATIONS.—Bones. Eyes (especially iritis). Acute nephritis. Nails. Acute myelitis. Joints. Testes. Effect on pregnancy.

Secondary lesions possess a general tendency to be symmetrical.

Rash.—

GENERAL CHARACTERISTICS.

1. POLYMORPHIC.—Macules, papules, etc., present simultaneously, yet spots tend to be of similar size; roundish; except roseola, are infiltrated.
2. ROUGHLY SYMMETRICAL. Abundant. On flexor rather than extensor surfaces. Occasionally a few spots only, e.g., on flexor surfaces of forearms.
3. COLOUR.—A coppery tint is specially suggestive.
4. DOES NOT ITCH.
5. Disappears without treatment.

May resemble any known rash, e.g., seborrhœa.

MAIN VARIETIES.

1. MACULAR SYPHILIDE, SYPHILITIC ROSEOLA. Commonest type and earliest onset. *Appears* about six weeks after chancre. *Duration*, three to six weeks. Rose-coloured spots, size about ¼ inch; when well developed, do not disappear on pressure; no infiltration. On trunk and flexors of arms; very rare on face. Leaves brownish stain. Recrudescence not uncommon, sometimes in late stages.
2. PAPULAR OR LENTICULAR SYPHILIDE. Onset tends to be later than previous type. Raised, often coppery, shiny, scales at margins, infiltrated. Desquamates. Abundant distribution, includes face. *Duration*, one to three months or longer.
3. PAPULOPUSTULAR SYPHILIDE.
4. SQUAMOUS SYPHILIDE (syphilitic psoriasis). Resembles psoriasis, but less silvery and scaly, infiltrated, and mainly on flexors: development rapid, often coppery tint, fissures common.
5. RUPIA (crusts form over ulcers). ECTHYMA (ulceration of pustules).—These are rare forms developing in neglected pustular eruptions.

DIAGNOSIS.—

PITYRIASIS ROSEA. — Diagnosis from macular syphilide. Itches: covered with fine scales: glands and mucous membranes unaffected. Scaly syphilides are infiltrated and less pink.

LICHEN PLANUS. -Diagnosis from papular syphilide. *Lilac tint*, flat-topped, polygonal, shiny. *Itches*.

PSORIASIS. Mainly on elbows and knees. Shiny and scaly. Chronic.

Sore Throat. --Tonsils swollen: *ulcers*, small, gray, clear-cut, shape (*a*) kidney or (*b*) 'snail track', often symmetrical Entire mucous membrane of mouth and tongue (glossitis) often inflamed: also *larynx* (hoarseness).

Mucous Patches. Flat gray areas *Site*, moist regions, especially angles of mouth, and also within mouth, e g, on tonsils

Condylomata. Papules, from hypertrophy of papillæ, moist, round. Very infective; always syphilitic *Sites*: skin surfaces in apposition, i e, external genitals, perineum, toes, under breasts Specially in women.

Lymphatic Glands.- *Generalized slight adenitis*, especially *epitrochlear* and *cervical glands* Never suppurate.

Alopecia. Hair loses gloss and falls out often in patches Grows again after treatment.

Anæmia. Very common. About 3000,000 red cells per c mm

Fever. —Usually slight Very rarely severe

Other Lesions, less frequent or characteristic

BONES - (1) Wandering (osteocopic) pains common mainly at night, (2) Symmetrical subacute periostitis of long bones frequent, effusion results in *nodes*, e g, on edges of tibia

EYES. *Lesions* common, usually in second year iris muddy, pupil small and reacts sluggishly Rarely, choroiditis and retinitis

NAILS 'Syphilitic onychia' ulceration around and decaying nail nails brittle

Occasional Lesions -

ACUTE NEPHRITIS Tends to be very severe. *See* RENAL SYPHILIS, p 272

ACUTE MYELITIS *See* SYPHILIS OF THE NERVOUS SYSTEM

JOINTS Very rarely affected in acquired syphilis Subacute painless symmetrical arthritis, usually knees

TESTIS Rarely affected. Epididymitis or orchitis.

Pregnant Woman usually aborts.

Late Secondary Syphilis. –

Certain symptoms tend to occur late in the secondary stage, or there may be recurrences of former symptoms. Emphasising that the division into stages is not absolute; they may even occur years after infection.

RASHES.—Any type of secondary rash may recur, especially

Syphilis—Late Secondary, *continued.*

roseola. Usually less characteristic in late stages. Rarely occasionally.

IRITIS.—Essentially a late secondary manifestation.

SUPERFICIAL GLOSSITIS.

ACUTE MYELITIS.

ORCHITIS.—Painless and symmetrical.

Some of these are variously regarded as late secondary or as tertiary manifestations.

Tertiary Stage.

Onset—Usually from two to ten years after infection. Occasionally after six months. No absolute upper limit.

Duration.—Unlimited. Recurrences common.

Tertiary manifestations result from chronic inflammation of cellular tissue, which may be (1) *Diffuse*, as in syphilitic cirrhosis of the liver; (2) *Localised*, viz., the *gumma*. Action on the blood-vessels, viz., various forms of arteritis, takes part in all lesions A classification follows, but the pathological basis is the same throughout, with the gumma as its predominant expression.

Lesions of Tertiary Syphilis.—

i. Gumma.

ii. Cutaneous and mucous-membrane lesions.

iii. Visceral lesions: (1) Nervous system. (2) Circulatory system: (a) Mesaortitis (aneurysm); (b) Obliterative endarteritis, etc. (3) Liver. (4) Testis (5) Bones. (6) Alimentary system (rectum). Rare: (7) Respiratory system, (8) Kidneys

iv. Various lesions: Miscarriages, effect on pregnancy. Amyloid disease.

Gumma.—No tissue or organ immune (except possibly prostate) especially in skin, mucous membrane, subcutaneous tissue, and muscles.

CLINICAL APPEARANCE (e g, in subcutaneous tissue) Firm, painless swelling develops rapidly, enlarges, softens, ruptures, discharges contents: ulcer results.

(1) ULCER.—Circular; deep, wall steep and 'punched-out', floor, yellow 'wash-leather slough'; base infiltrated, foul discharge common.

TERMINATION.—Varies with site and treatment. Responds rapidly to treatment except in brain.

(1) ABSORPTION.—With treatment, if gumma is unruptured, this may be practically complete (e.g., in testis). May be absorbed after fluctuation.

(2) ULCERATION, HEALING, AND SCAR.—*Thin 'tissue-paper' scar, usually pigmented.* Almost, but not quite, pathognomonic of syphilis.

ON BONES.—Hardens, producing osteosclerosis.

RECURRENCES.—Frequent.

SCARRING.—In certain sites may cause serious deformities, e.g., larynx, rectum, and liver.

DIAGNOSIS.—(1) Origin without cause, grows rapidly, softens, ulcer distinctive; (2) History and signs of previous syphilis; (3) Wassermann reaction positive; (4) Yields to antisyphilitic treatment. Spirochætes can rarely be found.

Cutaneous and Mucous-membrane Lesions.—

NODULAR CUTANEOUS SYPHILIDE (*tertiary or tubercular syphilide, syphilitic lupus*).—Commences as small brownish nodules, which enlarge; area increases by coalescence with fresh outlying nodules, producing serpiginous syphilide. *Margins* round or roughly crescentic; diameter 1 inch and upwards. At edges raised nodules Periphery extends, while in centre healing and scarring occur in various degrees. Skin thickens. *Site* usually single; especially forehead, neck, back, and scrotum, also palms and soles Resembles *lupus vulgaris*: distinguished by (1) rapid growth, (2) no apple-jelly nodules.

MULTIPLE CUTANEOUS GUMMATA.—Condition more severe than last: numerous gummatous ulcers

MUCOUS MEMBRANES Gummata common: ulceration very rapid; destroys all tissues, e g, nasal cartilage; heals with much scarring and deformity, whence strictures. *Sites*: nose; palate (perforations), larynx (strictures); pharynx; rectum (strictures); tongue, often with leucoplakia (*see* SYPHILIS OF TONGUE)

RARE CUTANEOUS CONDITIONS.—Leucodermia is sometimes syphilitic (or parasyphilitic) Keratodermia of the soles.

Visceral Lesions.—*See* p. 269 and elsewhere

Amyloid Disease.—Common in chronic syphilis

Effect on Pregnancy.—*See* CONGENITAL SYPHILIS

Quaternary Stage or Parasyphilis.

Diseases occurring usually many years after infection: (1) Tabes dorsalis, (2) Dementia paralytica. Syphilitic origin established by (*a*) Accumulated evidence of preceding syphilis, (*b*) Wassermann reaction, (*c*) Presence of *Spirochæta pallida* in tissues

CONGENITAL SYPHILIS.

General Principles.—*Inheritance of syphilis and effects on pregnancy*

1. EFFECTS OF SYPHILIS ON PREGNANCY.—*Repeated* miscarriages suggest syphilis The liability diminishes with the interval since infection, and with treatment. Typical results are: (1) *Repeated miscarriages*. (2) Waning effects in successive pregnancies—e g, sequence in 6 consecutive pregnancies: (i) early abortion, (ii) miscarriage in later months; (iii) syphilitic infant, death in few days; (iv) healthy at birth, syphilis in few weeks (typical 'congenital syphilis'); (v) malnutrition only, possibly interstitial keratitis later; (vi) healthy life.
2. CONGENITAL SYPHILIS.—Always inherited from mother, in whom Wassermann reaction is positive even if no symptoms are present: this explains Colles's law (*see above*).

Syphilis—Congenital, ***continued.***

3. CONGENITAL SYPHILITIC CHILD shows: (1) Wassermann reaction positive; (2) Immunity to acquired syphilis (at least until puberty)—this explains Profeta's law; (3) Response to treatment.
4. SYPHILITIC FATHER —Has syphilitic child only if his lesion can infect the mother.
5. TRANSMISSION TO THIRD GENERATION unproved.

Symptoms. –

A. PRESENT AT BIRTH, death occurring within a few days – Emaciated and feeble bullous eruption on palms and soles, syphilitic pemphigus neonatorum; snuffles, epiphysitis and disease of skull bones; enlarged liver and spleen Rarely, syphilis hæmorrhagica neonatorum

Syphilitic fœtus has large spleen and liver, teeming with spirochætes, bone changes, and various syphilitic lesions The placenta shows cirrhosis and arterial changes Hydramnios common

B APPEAR A FEW WEEKS AFTER BIRTH ('congenital syphilis') Healthy at birth Symptoms divisible into (1) Early symptoms (2) Late symptoms Both groups suggest long-drawn secondary stage, lesions similarly tending to be symmetrical (3) Tertiary and parasyphilitic lesions Any symptom of acquired syphilis may occur.

Early Symptoms.—

WASTING WITHOUT CAUSE MUDDY COMPLEXION

'SNUFFLES' – Onset three to eight weeks A syphilitic rhinitis, causing: (i) Contagious discharge, whence 'snuffling', (ii) Necrosis of nasal bones, whence later characteristic depressed bridge of nose

SKIN AFFECTIONS Onset three to twelve weeks (i) Roseolar rash: napkin area, may ulcerate (ii) Squamous eruption on palms and soles thickening with *profuse desquamation* (iii) Ulceration at angles of mouth ('rhagades'), whence later radiating scars, (iv) Hair loses gloss and falls out, especially eyebrows

ENLARGEMENT OF LIVER AND SPLEEN —May be jaundice

BONE AFFECTIONS —(i) *Syphilitic epiphysitis* ends of long bones often symmetrical or multiple. Occurs within first few months Rapid loss of movement (syphilitic pseudoparalysis) Epiphyses may suppurate or separate. Diagnose from rickets by (*a*) early age, (*b*) localization of thickening (ii) Bossing of frontal prominences of skull. (iii) Craniotabes (not confined to syphilis). (iv) Syphilitic dactylitis phalanges, metacarpals, metatarsals. From second year onwards Swelling may rupture Diagnosis from tuberculosis difficult

GENERAL GLANDULAR ENLARGEMENT is uncommon

OCCASIONAL SYMPTOMS – Iritis, onychia, various rashes, orchitis. Very rarely, hair becomes thick (syphilitic wig).

Progress.—Improves under treatment Development is slow: may be 'infantilism'.

Late Symptoms.—Onset during second dentition or puberty

EYE.—*Interstitial keratitis, bilateral* may cause blindness cornea steamy (ground glass); duration one to two years Prognosis good clears from periphery to centre, where opacities may remain Commonest late symptom may be sole syphilitic lesion *Iritis, disseminated choroiditis* not uncommon, often with keratitis, prognosis worse, vision permanently affected Rarely, optic atrophy

ARTHRITIS.—Painless, symmetrical, with effusion Usually in *knees* Arthritis of this type is always syphilitic

BONES Symmetrical periostitis, especially of *tibiæ* Result inflammatory thickening mainly in middle whence *sabre shaped curvature* Syphilitic dactylitis

EAR Causeless, rapid, permanent, bilateral deafness probably labyrinthine Age 11 to 20 years Females predominate

HUTCHINSON'S TEETH Upper central *permanent* incisors stunted, peg shaped, cutting edge smaller than base edge deeply notched, exposing dentine Rarely recognizable in adults owing to rapid erosion of cutting edge

Tertiary Lesions. Gummata not common but may occur as in acquired syphilis (especially testes

Parasyphilis.

JUVENILE GENERAL PARALYSIS Rare Occurs at about 16 years *Tabes dorsalis* is considerably rarer See SYPHILIS OF THE NERVOUS SYSTEM

Residual Symptoms. (1) Pallor malnutrition (2) Depressed bridge of nose (3) Radiating scars at angles of mouth (4) Square or asymmetrical skull (5) Liver and spleen may be palpable (6) Wassermann reaction positive (7) Thickening of tibiæ (8) Corneal opacities (9) Hutchinson's teeth

VISCERAL SYPHILIS

Syphilis of the Lungs.

Very rare Most important is the fibrosis at the root of the lung, the frequency of which is not yet fully known

1 **Congenital Syphilis.**—*White pneumonia of the fœtus*. large areas airless, gray, and smooth (not granular, as in 'gray hepatization') alveolar walls thickened filled with desquamated cells numerous *Spirochæta pallida* Pathological interest only life not exceeding few hours

B **Acquired Syphilis.**—

1 INTERSTITIAL PNEUMONIA (fibrosis) at *the root of the lung* fibrosis spreads outwards along bronchi and vessels May be associated with gummata and with bronchiectasis Characters: (i) Symptoms in general resemble pulmonary tuberculosis, tubercle bacilli absent, (ii) Changes mainly at root of lungs, noticeable in radiograph (iii) Improve with antisyphilitic

Syphilis of the Lungs, *continued.*

treatment; (a) Syphilitic history and other lesions may be present; (b) Wassermann reaction positive.

2. GUMMATA.—Very rare. Usually numerous and encapsuled: may be caseous and bronchiectatic cavities.

Syphilis of the Tongue.

Lesions frequent; some characteristic. Carcinoma may follow.

PRIMARY.—*Chancre.*

SITE.—Usually near tip on dorsum Indurated. Ulceration may be deep.

DIAGNOSIS.—(a) Epithelioma, at sides of tongue, painful. (b) Tuberculous ulcer, painful, pulmonary disease advanced.

SECONDARY.—Superficial glossitis.

TERTIARY.—

LEUCOPLAKIA.—Mucous membrane thickened and white, especially in smokers. Proof of syphilis not invariable. Carcinoma may follow.

SYPHILITIC GLOSSITIS.—Diffuse gummatous infiltration: results in deep fissures, large hard tongue, leucoplakia common Very characteristic.

LOCALIZED GUMMATA.—Infrequent.

HERPES common.

Syphilis of the Liver. (*Syphilitic Hepatitis*).

A. Congenital Syphilis.—

1. DIFFUSE HEPATITIS.—Occurs in infants born with disease, or developing signs within a few weeks Present in most early fatal cases.

MACROSCOPIC.—Liver large and tough, of yellow or flinty colour.

HISTOLOGY.—Pericellular cirrhosis. Spirochætes in enormous numbers. (In early stages in fœtus, a diffuse, small round-cell infiltration)

PHYSICAL SIGNS.—Liver enlarged, below navel. Spleen also enlarged. Ascites rare. May be jaundice

2. LATER CONGENITAL SYPHILIS.—Liver changes similar to acquired forms

B. Acquired Syphilis.—

SECONDARY STAGE.—Jaundice occasionally probably catarrh of ducts.

Very rarely, acute (yellow) atrophy of liver and cholæmia, may occur in absence of salvarsan treatment.

TERTIARY STAGE.—Lesions important. (1) Gummata; (2) Scarring of liver. May co-exist.

GUMMATA.—*Size*: from a pea to a fist or larger. Often multiple. *Site*: any part, most commonly anterior surface, junction of right and left lobes. *Appearance*: firm, grayish, roughly spherical. Three zones present in early gummata, especially when large. (a) Caseous centre; (b) A surrounding

fibrous-tissue zone; (iii) Outer zone of small round-cell infiltration, where condition is advancing. *Progress*: caseation; then absorption partial or complete, resulting in *scarring*. Rarely softening or calcification. Local peritonitis may occur.

SCARRING OF LIVER Depressed scars on surface of liver: fibrous-tissue strands run inwards; may be gummatous or caseating areas Scarring of all degrees from small superficial linear scars up to extreme deformities (botryoid liver).

AMYLOID DISEASE —Now rare Other organs also affected.

SYMPTOMS —Two principal groups —

(i) TUMOUR OF THE LIVER (*gumma*) — Palpable mass: liver usually large and tender from perihepatitis. Pain in right hypochondrium or epigastrium common. *Spleen* may be palpable. Often no other syphilitic signs. *Diagnosis* from neoplasm difficult. *Wassermann reaction* positive Antisyphilitic treatment effective

(ii) RESEMBLES ATROPHIC CIRRHOSIS OF LIVER (*scarring*).— Fever and ascites may be jaundice Liver edge irregular, if palpable Portal obstruction probably mechanical from gumma or scarring in the portal fissure May be no syphilitic signs Hæmatemesis unusual.

Jaundice is sometimes the only symptom

Occasionally condition resembles hypertrophic cirrhosis of the liver, Banti's disease, splenic anæmia Rarely, symptoms suggest suppuration.

Syphilis of the Alimentary Tract.

Syphilitic lesions are very rare between the pharynx and the rectum

STOMACH.—Syphilitic lesions very rare at autopsy. The occurrence of a syphilitic gastritis and gastric ulcer is in dispute

INTESTINES — Lesions very rare Stenosis has resulted from gummata

SYPHILIS OF RECTUM —Not uncommon almost invariably women, probably direct infection from vulva and neighbourhood A slow gummatous growth immediately above internal sphincter; usually surrounds rectum

Stricture of rectum subsequently develops may be extreme, distinguished from neoplasm by hard fibrous ring.

SPLEEN —Enlargement not uncommon Gummata and scarring not infrequent; liver usually involved also

Syphilis of the Circulatory System.

THE HEART —The principal effects on the heart are due to syphilitic lesions of the coronary arteries and blood-vessels, resulting in fibroid myocarditis, and to syphilitic lesions of the aorta, resulting in aortic valvular disease.

Gummata are very rare, but some recorded cases are of special interest owing to their position in the bundle of His and functional tissues of auricle and ventricle, producing disturbances of cardiac rhythm and Stokes-Adams' syndrome.

Syphilis of the Circulatory System, *continued.*

THE ARTERIES. —*See* ARTERIOSCLEROSIS and ANEURYSM (With the exception of a small group in elderly persons, and a few rare congenital abnormalities, aneurysm is invariably of syphilitic origin.)

Renal Syphilis.

A. **Secondary Syphilis.** —

MILD SIMPLE ALBUMINURIA Not uncommon Prognosis good. Formerly ascribed to mercury, erroneously

ACUTE SYPHILITIC NEPHRITIS Of importance, owing to severity.

FREQUENCY About 4 per cent

ONSET Commonest two to four months after chancre, viz., at time of rash

SYMPTOMS Similar to ordinary acute or subacute nephritis, noticeable being (i) Albumin often in very large quantities may be 5 per cent, (ii) Severity of symptoms Mortality considerable, but after recovery chronic nephritis is rare

TREATMENT As in acute nephritis In addition, salvarsan preparations Mercury in moderate doses not contra indicated.

B. **Tertiary Syphilis.** Little importance.

1 Amyloid disease
2 Gummata of the kidneys very rare Undiagnosable
3 Interstitial nephritis Only with arterial disease

Syphilis of the Nervous System

See DISEASES OF THE NERVOUS SYSTEM

DIAGNOSIS OF SYPHILIS.

General Diagnosis. —Lesions often distinctive and simplified by multiplicity. History and signs of earlier disease often present, viz. —

PRIMARY CHANCRE Scar may be present Difficulties arise from absence of scar, urethral and extragenital chancres, masking by gonorrhœa or soft sore, and in females presence on os uteri

SECONDARY LESIONS Inquire and examine for these

TERTIARY LESIONS Examine for results of gummata, e.g., 'pigmented tissue-paper' scars on legs, perforations of palate, etc

IN WOMEN Repeated miscarriages.

CONGENITAL SYPHILIS —Residual phenomena (*see* p. 269), especially depressed nose, radiating scars from mouth, history of interstitial keratitis and blindness at puberty

Specific Diagnosis.—

SPIROCHÆTA PALLIDA In chancres, condylomata, and mucous patches. For methods, *see* 'Parasite', p. 162.

WASSERMANN REACTION.

CEREBRO-SPINAL FLUID

THERAPEUTIC TEST.—Response to antisyphilitic treatment. Not reliable.

A simpler test to replace Wassermann reaction is being searched for—e.g., Sachs-Georgi precipitation test, Vernes' test, Dreyer and Ward's 'Sigma' test. No one of them at present is proved to be equally reliable.

Wassermann Reaction. Performed on blood serum, or cerebrospinal fluid. The blood test is here particularly referred to.

POSITIVE REACTION.—*Is proof of syphilis* except that it also occurs in certain tropical diseases: leprosy, yaws, trypanosomiasis and in scarlet fever (unusual and transient).

Note.—A positive reaction does not prove that a given lesion, e.g., tumour of liver, is necessarily syphilitic.

DOUBTFUL REACTION.—Liable to occur, especially if patient is under treatment (a fact which may be concealed). Repeat—if necessary, after provocative dose of '606'.

NEGATIVE REACTION.—Value depends on such circumstances as lesion, e.g., chancre, treatment, etc. In absence of knowledge of treatment, does not negative syphilis.

Wassermann Reaction of Serum in Diagnosis—

PRIMARY STAGE (chancre).—Reaction becomes positive four to eight weeks after infection, viz. later than chancre; hence negative reaction no guide; but if negative at two months, without treatment, excludes syphilis.

SECONDARY STAGE.—Practically always positive.

TERTIARY STAGE.—Practically always positive and remains while symptomatic; active in 1 in 1,000 is always positive. With small lesions, many years after infection, is negative occasionally.

CONGENITAL SYPHILIS.—Always positive until puberty, and resistant to treatment.

LATENT SYPHILIS.—Previous syphilis but no symptoms: positive in 30 to 40 per cent. Mothers of syphilitic children but without symptoms, usually positive.

SYPHILIS OF NERVOUS SYSTEM.—Negative reaction does not exclude syphilis.

PARASYPHILIS.—*Dementia paralytica*: always positive (also cerebrospinal fluid). *Tabes dorsalis*: positive in over 50 per cent (indication for syphilitic treatment).

Cerebrospinal Fluid. In diseases of nervous system, examine cerebrospinal fluid for (1) Cells, (2) Globulin, (3) Wassermann reaction. Lange's gold test is not so reliable.

CELLS.—Small lymphocytes present. Over 10 cells per c.mm. is pathological. This is diagnostic in chronic conditions, but also occurs in tuberculous meningitis, and to some extent in acute poliomyelitis, encephalitis lethargica, and in chronic forms of cerebrospinal meningitis.

GLOBULIN.—Special tests. Not proof of syphilis.

WASSERMANN REACTION.—*Tertiary syphilis*, may be positive; *dementia paralytica*, always positive; *tabes dorsalis*, often positive.

Syphilis, ***continued.***

TREATMENT OF SYPHILIS.

Treatment must be commenced immediately, but not before, the diagnosis is established by unmistakable signs, or by presence of *Spirochæta pallida*, or positive Wassermann reaction. A general scheme is given first, and then individual drugs are referred to.

General Scheme of Treatment.—A patient should be watched for at least two years, and treatment is frequently necessary, intermittently, for this period or longer.

Treatment must include injections of both mercury and some arsenobenzol preparation. Numerous methods are in use, but general principles are fairly similar,* viz.: (1) Salvarsan, at least six injections, with total of 2·5 to 3 grm., commencing with 0·3 grm. (2) Mercury, at least 6 and preferably 9 or more injections of 1 gr. (3) Interval of one week between injections. (4) Period of injections not to exceed three months. (5) At conclusion of above, an interval of one month, then 3 or 4 injections of salvarsan or mercury. '914' may advantageously be used, the total dosage being half as large again as '606'. Mercury may be given at same time as arsenobenzol preparations.

Subsequent procedure depends on symptoms, Wassermann reaction, and cerebrospinal fluid. If Wassermann reaction was positive at onset, the entire course should be repeated. If in addition, active symptoms were present, short courses of injections should subsequently be given at intervals of two months.

SYPHILIS OF CENTRAL NERVOUS SYSTEM.—General principles: (1) Initial injections of mercury (2 or 3). (2) Commence salvarsan with small doses. (3) Give total of 4 to 5 grm. with weekly injections of mercury. Commence salvarsan with 0·2 grm. bi-weekly. Repeat entire course after two months' interval. A third or fourth course may be necessary. Control with Wassermann reactions, blood and cerebrospinal fluid and cell examinations in latter.

PREGNANT WOMEN.—Salvarsan and mercury until one month before labour.

MERCURY BY THE MOUTH. Subsequent course. If symptoms marked, or commencement of treatment at some interval from onset, mercury by mouth advisable, following above course: hydrarg. c. creta gr. j to ij, pulv. ipecac. co. gr. j, t.d.s. for two years. Omit for one month before Wassermann tests.

FOR GUMMATA AND TERTIARY LESIONS.—Potassium iodide gr. x, t.d.s.

Wassermann Reaction.—Test at end of course, and at three subsequent intervals of three months. If reaction positive or partial, renew course of injections, e.g., salvarsan preparation; three mercury injections; second salvarsan injection. Duration of course guided by strength of reaction. Repeat series of tests. *If the three tests are negative,* no further treatment necessary.

* *See* especially Harrison, *Treatment of Venereal Disease.*

(A 'provocative' half-dose injection of a salvarsan preparation is sometimes given and serum tested shortly afterwards, but this is not essential.) Wassermann reaction to be repeated after further six months, finally at two years from commencement of treatment. If positive, treatment to be renewed, guided by severity of reaction. If negative, disease may be regarded as cured.

NOTES:—

Correct treatment, especially in early stages, may temporarily convert a positive reaction into negative before the disease is cured.

Syphilis is not cured while reaction remains positive.

If a reaction initially positive becomes *persistently negative* for two years, cure may be supposed.

Arsenobenzol Preparations.

Salvarsan ('606'), dioxy-diamido arsenobenzol dihydrochloride, introduced by Ehrlich in 1909. Often known as arsphenamine. Identical preparations: *Kharsivan, Arsenobenzol* (*Billon*), etc.

PREPARATION FOR INJECTION.—Convert into sodium salt and make into dilute solution isotonic with blood. Solutions necessary: (1) Pure sterile *freshly distilled water*. (2) NaCl solution 0·5 per cent. (3) NaOH solution: the amount of this necessary is often stated on the preparation: also the strength, either 15 or 4 per cent. All solutions and vessels to be carefully sterilized and to be cold. Preparation: (*a*) In large sterile bottle place 10 c.c. water for each 0·1 grm. of '606'; add drug; shake until *completely dissolved* (aided by glass beads). (*b*) Add NaOH solution drop by drop until clear; precipitate forms and redissolves. (*c*) Add NaCl solution, 30 c.c. for each 0·1 grm. To be used within 30 minutes of preparation.

METHODS OF INJECTION.—*Intravenous* (no other route should be employed). median basilic or other convenient vein.

1. GRAVITY.—Glass funnel with rubber tubing and needle. Tubing filled with physiological saline solution: needle inserted into vein; '606' poured into funnel and allowed to flow; conclude with some physiological saline. All apparatus to be sterilized.
2. PRESSURE. Special apparatus.

Neosalvarsan ('914').—Identical are: *Neokharsivan, Novarsenobenzol* (*Billon*), *Novarsenobillon*. Also known as neoarsphenamine.

MODE OF INJECTION.—Dissolve drug in 10 c.c. cold sterile distilled water. Draw into 10-c.c. sterile syringe, place needle in position. Insert needle in vein and inject solution. To be used within 30 minutes of preparation.

Comparison of '606' and '914'.—'914' is readily soluble in water, and solution is neutral, hence has advantage of *great simplicity* of preparation and injection, of use in small amounts of fluid; and also is less toxic. '606' is regarded as slightly more efficacious by some authorities, but this is doubtful, and the above advantages of '914' are obvious.

Syphilis —Treatment —Arsenobenzol Preparations, *continued.*

Similar Preparations.—Numerous drugs are under trial, e.g., silver salvarsan (results good), luargol and disodoluargol (contains antimony and silver), sulphoxylate '1495'. Galyl is less effective.

Contra-indications.—Addison's disease and hæmophilia are absolute contra-indications. The following are indications for caution, e.g., small doses, previous treatment with mercury injections: Advanced visceral disease—*renal* (except when syphilitic), hepatic, myocarditis, arteriosclerosis, aneurysm, aortic disease, diabetes, alcoholism, and disease of *central nervous system.* Old age and infancy. Dementia paralytica is definite counter indication. In pregnancy with care.

Ill-effects.—Result from: (1) *Water*: prevented by fresh and careful distillation. Little liable to occur in small amount necessary for '914' and similar preparations. (2) Disease, as above. (3) Varying susceptibility. (4) *Herxheimer Reaction*: Administration of any powerful syphilitic remedy may cause transient exacerbation of symptoms, ascribed to destruction of spirochætes and release of toxins. Well seen in skin and mucous membrane lesions. Also Wassermann reaction becomes more positive. Obviated by alternating mercury with salvarsan. In advanced untreated secondary syphilis, give mercury as preliminary to salvarsan.

Sequelæ of Injections.—May be—

SYNCOPE.—During injection. Psychical. No importance.

PYREXIA, RIGORS, HEADACHE.—Within few hours, mainly from the water. Rare with careful preparation of water, and with '914'.

'NITRITOIDISM'—'ANAPHYLACTOID CRISES'.—Flushing of face, rapid pulse, dilated pupils. If severe, swelling of face, unconsciousness, twitching of limbs. Are vasomotor phenomena and not of anaphylactic origin. Give injection intramuscularly, 5 min. of 1-1000 adrenalin hydrochloride or injection of atropine. Purgative.

SKIN ERUPTIONS.—*Urticaria*, especially after nitritoidism. *Erythema*, very rarely progresses to exfoliative dermatitis.

VOMITING AND DIARRHŒA.—Rarely severe.

HERXHEIMER REACTIONS.—*See above.*

CRANIAL NERVE PARALYSIS.—Usually 7th or 8th nerve. Rare. From *over-small dose.* Repeat dose. Prognosis good.

SEVERE CEREBRAL SYMPTOMS.—Convulsions, coma, etc. The cause of most fatalities. Very rare. Employ venesection.

JAUNDICE.—May, very rarely, progress to acute yellow atrophy.

NEPHRITIS.

Arsenobenzol preparations damage endothelium of blood-vessels, which may result in capillary thrombosis and minute hæmorrhages: coma is thus produced, also hæmorrhagic nephritis, and petechiæ, and very rarely purpura.

The vast majority of fatalities occurred in the early days of injections, and were due to faulty technique now extremely rare

Preparation of Patient. – Aperient on previous evening Exclude albuminuria. Rest for a few hours after injection

Effects of Arsenobenzol Preparations. – (a) Spirochætes in local lesions disappear in twelve to twenty-four hours, (b) Lesions improve rapidly For curative treatment, combine with mercury administration Relapses few Wassermann reaction becomes negative in early cases In syphilis of the central nervous system and tabes dorsalis action slighter, but should be repeated whenever symptoms recur *Dementia paralytica is a definite contra indication*

Mercury.

Mercury alone has cured syphilis in the past but needs prolonged course, and relapses are common Best treatment is course of weekly intramuscular injections (Lambkin's cream), combined with 606 or 914' Mercury nowadays must not be relied upon alone

Remove teeth before treatment Cleanse mouth frequently Smoking only in moderation

SIGNS OF OVERDOSE OR INTOLERANCE Salivation Sore gums, Gastritis. Diarrhœa

METHODS OF ADMINISTRATION

INTRAMUSCULAR Rapidly administered Dose certain

INUNCTION Absorption rapid but variable, and needs conscientious application Useful in infants

BY MOUTH Course very prolonged Most useful following intensive course of treatment

Intramuscular Injections. Preparations employed may be (a) Insoluble (b) Soluble

PREPARATIONS

INSOLUBLE Lambkin's mercurial cream gr. j in ℿx heat bottle to blood heat thoroughly Or olive oil Dose gr. j mercury or calomel One injection every 7 days About five injections

SOLUBLE Mercuric iodide, 1 per cent solution in oil Dose 0.5 to 1 c.c One injection every 7 days Thirty to forty injections Action more rapid than insoluble preparations

METHOD OF INJECTION – Complete asepsis Site Buttock, measure line from anterior superior spine of ilium to upper end of interglutæal fold inject at junction of outer and middle third. Needle thrust 1½ inches deep

Inunction.—Ung hydrarg ʒj daily for adult Rub in for 20 minutes Thirty to forty inunctions Avoid hairy sites, and vary daily Miss every seventh day For infant, 10 to 30 gr

Contra-indications Pustular syphilides. Delicate skins

By Mouth. –Hydrarg c crêta gr. j to ij, pulv ipecac co gr. (Hutchinson's pill), t d s. for two to three years. Or liq. hydrarg.

Syphilis—Mercury, *continued.*

perchlor. ʒss to ʒj, t.d.s. In tertiary lesions or relapses can be given with pot. iod.

Potassium Iodide.

In gummata and tertiary lesions.

ACTION.—Removal of chronic inflammatory tissue. No direct effect on spirochætes.

ILL EFFECTS.—Coryza and conjunctivitis. 'Iodide rash' pustular eruption. Disappear on remitting drug.

DOSAGE.—Gr. v to x, t.d.s., with ammonium carbonate, e.g.—

℞ Pot. Iod. gr. v; Ammon. Carb. gr. ij; Aq. Menth. Pip. ad ʒss

Special Treatment.

CHANCRE.—Wash. Local applications: dermatol; lotio nigra (add tr. opii ʒj in ℥j, if painful). Excision of a chancre appears to be useless.

PHAGEDÆNA.—Continuous bath.

RASHES.—If obstinate, Donovan's solution ♏v, t.d.s.

CONDYLOMATA.—Dry. Starch and calomel dusting powder.

CHAPTER XLVI.

YELLOW FEVER.

An infectious disease due to a spirochæte, transmitted by the mosquito *Stegomyia fasciata*, and characterized by jaundice, albuminuria, hæmorrhages, especially from the stomach ('black vomit'), and slow pulse with a rising temperature. The course shows three stages: Continued pyrexia; Remission; Secondary fever.

Etiology.—White races more susceptible than coloured. One attack protects. Frequent in late summer months. Epidemics cease with cold weather.

Morbid Anatomy.—The general condition is a severe acute general toxæmia, and the lesions are not specific.

SKIN.—Jaundiced. Subserous hæmorrhages usual.

STOMACH.—Contains fluid black blood. *Submucous hæmorrhages* in stomach and duodenum; ileum usually free.

LIVER.—Invariably advanced changes of acute necrosis and fatty degeneration: substance yellow and friable: condition of 'icterus gravis'.

KIDNEYS.—Acute nephritis, in varying degree.

LUNGS.—Congested, hæmorrhages common.

SPLEEN.—May be enlarged.

BLOOD.—Serum may contain hæmoglobin; urea in blood often enormously increased.

Fatty degeneration of heart, kidneys, etc., may be present.

Mode of Transmission.—The work of the American Commission has shown conclusively :—

i. The mosquito *Stegomyia fasciata* ('tiger mosquito') conveys the infection after feeding on blood of infected persons. No other mosquito can do so. Mosquito remains infective about sixty days or as long as it lives. This is the sole method by which the disease is transmitted in nature. *Cases of yellow fever* carried to places free of *Stegomyia fasciata* are incapable of spreading infection.

ii. *An infected mosquito* after feeding cannot transmit infection until an interval of about twelve days. Thus organism is not merely transmitted, but must pass through some developmental stages, as in malaria.

iii. *Blood of infected persons* contains the infective agent in first three days only of the disease, and reproduces disease on inoculation of 1 c.c. or less subcutaneously.

iv. The fomites, vomit, fæces, etc., and clothes of infected persons *never convey infection.*

v. *Virus* passes a filter (Berkefeld and Chamberland F, but not Chamberland B). Destroyed by heating to 55° C. for ten minutes.

MILD TYPES occur in children in infected districts, and form reservoir of infection.

Bacillus icteroides, Sanarelli, 1897, is frequently present in fæces in later stages, as a secondary invader.

VIRUS.—A spirochæte, *Leptospira icteroides*, identified by Noguchi, 1919. Isolated by injecting patients' blood into peritoneal cavity of guinea-pigs. Is agglutinated by serum of convalescents. Can be cultivated. Occasionally recognized in patients' blood. An antiserum has been prepared. Closely related to spirochæte of Weil's disease.

EPIDEMICS. Commence about two weeks after primary case, i.e., twelve days for development in mosquito and two to five days incubation period in man.

DISTRIBUTION.—Endemic and epidemic in West Indies and Atlantic Coast from New York to Rio de Janeiro; also in West Africa. Occasionally imported into Europe and sporadically into England. Ships were specially liable to epidemics in the past, and to convey infection. *Stegomyia fasciata* keeps to low altitudes, near sea coast and big rivers, and thus determines distribution. Eggs are laid in water, but are resistant even to drying. Young mosquitoes, which only feed by day, are rarely infected. Old mosquitoes feed in evening and early morning, and never in the middle of the day, at which time risk of infection is consequently almost negligible.

DURATION OF INFECTIVITY.—Not exceeding four days.

QUARANTINE PERIOD.—Six days. None for 'immunes' (previous attack).

Yellow Fever, *continued*

Symptoms.—

INCUBATION PERIOD.—Three to four days. Experimental limits, two to six days.

ONSET.—*Sudden*, with rigor, often in early morning. Usual early symptoms: severe headache, often frontal, pains in back, rapid pyrexia, skin dry. Three stages are often distinctly marked.

STAGE 1.—*Continued Pyrexia.*—Duration one to three days. Temperature high from onset, 100° to 106°, remains steady or rises. Three important symptoms are:—

a *Facies*: face flushed, eyes red and injected with definite icteroid tinge.

b *Pulse-rate* commences to fall with steady or with rising temperature.

c *Albuminuria* on third day. Urine scanty.

Other symptoms:—

Vomiting: first of food, then of acid and blood towards end of period. *Constipation*. Headache. Epigastric pain. *Pains* in body and limbs of varying severity, may be intense.

STAGE 2.—*Stage of Calm or Remission.*—Duration one to three days. Change from previous stage rapid. Temperature falls nearly to normal, pulse slows further and symptoms diminish. In mild cases convalescence may now set in. Rarely, anuria and black vomit occur: death then almost invariable.

Serious cases more frequently pass into next stage.

STAGE 3.—*Secondary Fever.*—Onset about fifth day. Duration a week or more. This is the critical period. (i) Temperature rises gradually to 104°. (ii) Pulse-rate continues to fall. (iii) Jaundice intense. (iv) Vomiting recurs ("black vomit"). (v) Urine diminishes: albuminuria increases. Abdominal pain and melæna. Prostration and weakness extreme.

In favourable cases symptoms commence to subside gradually about eight days from onset.

In unfavourable cases (i) Vomiting and jaundice increase, or (ii) Suppression of urine occurs, with delirium, uremic convulsions, coma, and death. Groups (i) and (ii) often occur together.

Summary of Symptoms.—

JAUNDICE.—Icteroid tint in conjunctivæ at onset. Jaundice becomes extreme.

TEMPERATURE.—(1) In first stage high from onset, usually 103° to 105°; steady or rises. (2) In second stage falls to 98° to 100°. (3) In third stage rises to 101° to 103°: in favourable cases after about three days commences to fall by lysis: but in unfavourable cases usually rises continuously until death.

PULSE-RATE.—On first day 100 to 110, then falls to about 75 at end of first stage. Subsequently falls lower to about 50. *The falling pulse-rate with steady or rising temperature is characteristic* (Faget's sign).

URINE.—Onset of albuminuria usually on third day, even in mild cases. In second stage may be absent. Then returns, with presence of casts, blood, bile, and changes as in acute yellow atrophy. In third stage anuria frequent, with uræmia and rapid death.

VOMITING. In first stage, nausea and vomiting of food, acid, and blood. In third stage, 'black vomit', black fluid containing blood pigment. Amount very large. Emesis with effort Epigastric pain severe.

SPLEEN. Not enlarged except with malaria.

CONSTIPATION. Stools not clay coloured until late. May be 'tarry' from blood

MENTAL CONDITION May remain clear. Delirium in severe cases.

PETECHIÆ May develop on skin and mucous membranes.

BLOOD. No leucocytosis. Bile present may be free hæmoglobin.

Varieties of Types.—

MILD. Symptoms of onset and first stage only. No black vomit. Duration two to three days Diagnosis difficult except in epidemics. Probably forms a considerable proportion of cases

SEVERE. Petechiæ on skin, hæmorrhages from mucous membranes, hæmaturia, etc

MALIGNANT. Second stage usually occurs, and patient dies from toxæmia, with very slight symptoms.

Progress. *Relapses* are uncommon *Sequelæ* are rare occasionally boils or diarrhœa *Convalescence* usually surprisingly rapid, and strength returns quickly.

MORTALITY In white patients under good conditions should not be more than 10 to 15 per cent High mortality among alcoholics and debilitated subjects

Diagnosis. In epidemics simple The important diagnostic symptoms are (1) Early intermediate tint development of deep jaundice, (2) Falling pulse with steady or rising temperature, (3) Albuminuria; (4) Black vomit Diagnosis from

BLACKWATER FEVER Similar symptoms are jaundice, vomiting, suppression of urine In blackwater fever, no headache, no falling pulse, hæmoglobinuria constant, hæmatemesis very rare. Hæmoglobinuria rare in yellow fever, though hæmaturia not uncommon

MALARIA. Protozoa in blood; enlarged spleen, no early jaundice.

DENGUE.—Difficulty occasionally caused by coexistence

RELAPSING FEVER. Similar symptoms are jaundice, vomiting; rarely black vomit. In relapsing fever. *Spirilla obermeieri* present in blood, also enlarged spleen.

Prophylaxis. The American Commission in Cuba and Panama have shown the possibility of stamping out yellow fever.

Yellow Fever—Prophylaxis, *continued.*

1. Prevent breeding of mosquito, by destruction of breeding-places.
2. Screen patient's bed with mosquito nets, to prevent infection of mosquitoes.
3. Fumigate houses of patients in order to kill infected mosquitoes.

Treatment.—

GENERAL TREATMENT.—Bed. Bland fluid freely, especially soda-water with sodium bicarbonate, gr. lx to the pint; all fluid to be given cold. No food for three or four days. Alcohol advisable. *At onset,* dose of calomel followed by saline purge, but do not repeat. Enema when necessary.

STERNBERG'S 'ALKALINE TREATMENT'.—Gives good results. Sodium bicarbonate, gr. vj, in three tablespoonfuls of water hourly, with calomel gr. $\frac{1}{10}$.

FEVER.—Treat by hydrotherapy (*see* TYPHOID FEVER) A simple diaphoretic mixture may be given.

VOMITING.—Ice to suck and ice to stomach. Champagne. All fluids in small quantities. If necessary, rectal salines for 'black vomit'.

URÆMIA.—Usual methods Treatment of little value Note The urine should be measured daily

HEART FAILURE.—Alcohol, digitalis, ether.

QUININE.—Is useless in treatment.

Experiments with antisera are in progress.

CHAPTER XLVII.

BRONCHOPULMONARY SPIROCHÆTOSIS.

(*Spirochætal Bronchitis*)

Described by Castellani in Ceylon in 1905 Apparently universal distribution.

Spirochæte.—Markedly pleomorphic; great variations in length and breadth. Actively motile in fresh specimen, but shortly assumes a coccoid form, a resting stage. Stains readily with ordinary stains. By most authorities considered a distinct species from mouth spirochætes.

Clinical Characteristics.—

1. ACUTE.—Pyrexia 103°. Cough. Sputum scanty, mucopurulent —rarely trace of blood. General pains. Duration: few days, rapid improvement.

2. SUBACUTE.—Onset sudden or gradual. Often little or no fever or general disturbance. Cough frequent. Sputum blood-stained, or definite hæmoptysis. Physical signs: bronchitis; may be patch of consolidation. Duration: two to several weeks.

3. CHRONIC.—Onset insidious, or less often sequel to above. Resembles chronic bronchitis, but sputum often sanious, or definite hæmoptysis. May closely simulate tuberculosis, viz.: irregular pyrexia, wasting, crepitations, and dullness in lungs. Course: chronic with remissions; hæmoptysis may be prominent.

Diagnosis.—By spirochætes in sputum (wash mouth carefully). From chronic bronchitis, and especially tuberculosis.

Treatment.—Arsenic, e.g. liq. arsenicalis. Arsenobenzol preparations often act as specific.

Caisson Disease, *continued*

During compression, viz., under the high atmospheric pressure of the occupation, the tissues absorb much nitrogen from the blood. The amount absorbed varies with: (1) The *pressure* Each 33 feet of water adds one atmospheric pressure (14·7 lb. per sq inch) (2) The *duration of exposure.* In about 1½ hours the tissues are practically fully saturated for the particular pressure present. (3) *Muscular work* Increases rapidity of absorption.

During subsequent decompression, viz, return to normal pressure, tissues pass into condition of supersaturation, and must part with nitrogen in order to establish equilibrium with the surrounding air If decompression be sufficiently slow, the blood can remove the excess and discharge it through the lungs If decompression be too rapid, bubbles of nitrogen gather in the tissues and produce symptoms mechanically by their presence.

Factors in this phenomenon are.—

1. The higher the pressure, the longer the working shift, and the shorter the period of decompression, the greater is the risk (Leonard Hill).
2. The brain and cord are practically in closed cavities further, the spinal circulation is slow Hence the affection of the nervous system
3. Fatty tissue absorbs much nitrogen

Divers go to greater depths than caisson workers, but for shorter periods and lighter work, and hence are less affected Record is 210 feet. *Tunnel workers* are also under compressed air

Morbid Anatomy.—Laceration in spinal cord Congestion of nervous system and internal organs May be much gas in heart-blood: analysis has shown 82 per cent N In chronic cases, may be typical chronic myelitis

Symptoms.—

ONSET.—Usually half to one hour after decompression

MILD FORMS.—Headache, giddiness, faintness transient

SEVERE FORMS.—(1) *Agonizing pains*, chiefly legs and abdomen (the bends') (2) *Paralysis*. rapid onset, usually legs and abdomen. both sensory and motor All degrees. Occasionally headache, giddiness, vomiting

EXTREME FORMS.—Unconsciousness of apoplectic character Rapid death.

Progress.—

RECOVERY.—Usual

PARALYSIS.—(*a*) May recover in few days even when complete or gradually; (*b*) Permanent, similar to transverse myelitis.

RAPID DEATH.—In cases severe from onset

Prognosis also varies with: (*a*) Age (50 years should be maximum age in such occupations); (*b*) Condition of heart, (*c*) Degree of adiposity.

Prophylaxis.—Gradual decompression, affording time for escape of nitrogen. Two principal methods:—

a. Leonard Hill.—Decompress at pressure of 20 lb. for a period which allows 20 minutes for each atmosphere present during occupation.

b. Haldane and Boycott's 'Stage Method'. -Decompression in several stages, at various pressures, for periods *increasing* as pressure approaches normal.

Both methods are effective, cases of disease only rarely occurring. Hill's method is simplest. To prevent *cardiac exhaustion*, no shift at high pressures should exceed two hours. During decompression, escape of nitrogen is aided by muscular movements and by a high percentage of oxygen.

Treatment.- If symptoms commence, subject must be recompressed in a medical air-lock, and very slowly decompressed.

EFFECTS OF CURRENTS OF ELECTRICITY.

Strength of Current. Current of 500 volts is usually fatal. Lower currents, even 120 volts, may be fatal *Alternating* currents are *more dangerous* than direct currents.

Grip cannot be relaxed when grasping a current of 50 volts with wet, or 100 volts with dry, hands. Current of 65 volts has been fatal, and one of 6000 volts non fatal (Oliver) A current of 1500 volts is used in America for electrocution.

EFFECTS.—Vary greatly with (1) *Moisture of skin*, and degree of insulation, e g, wet boots; (2) Duration and completeness o contact; (3) General health or debility.

ACTION. (1) High currents (over 1200 volts) inhibit nervous centres; (2) Lower currents are fatal by causing fibrillation of the heart; (3) Spasm of muscles, (4) Burns.

Morbid Anatomy. Changes slight. Capillary hæmorrhages and congestion of nervous system occasionally. Blood usually fluid; reduced hæmoglobin present. Burns may be present.

Symptoms.—

NON-FATAL

MUSCLES. With passage of current, muscles contract in tetanic spasm. hence a grip cannot be relaxed until circuit is broken. Results in (1) great pain, (2) terror.

SYNCOPE.—Common: usually short duration.

SEQUELÆ may be:—

(1) Hysteria and traumatic neuroses: common, from terror or shock.

(2) Hemiplegia and other organic paralyses: very rare, but apparently authentic.

(3) Visual disturbances.

FATAL.—Severe spasm of all muscles, may throw body some distance from source of current: often a cry. (1) Death instantaneous, high currents inhibiting nerve centres; (2) Unconsciousness, heart ceases, death in three to four minutes.

BURNS.—May occur either in fatal or non-fatal cases. Vary greatly with moisture of skin and duration of contact.

Effects of Currents of Electricity—Burns, *continued.*

CHARACTERISTICS —

Skin blackened and dry. Never moist. Never suppurates.

Painless, but surrounding tissues often tender.

Loss of substance usually slight.

Heal very slowly. Burn may affect all tissues to bone. Gangrene may occur.

Treatment. -

If the body is in contact with the current. Preferably switch off current. If impossible, kick body away, or use hands covered with rubber gloves or with dry cloth. (*Beaute de mieux*, a man may remove his coat and, holding the inner side before him, pass his hands half way down the sleeves.) Never use bare hands.

If patient is unconscious. Artificial respiration, *to be persisted with for at least two hours.*

Section III.—THE INTOXICATIONS.

CHAPTER XLIX.

ALCOHOLISM.

The effects of excess of alcohol are here considered in groups (1) *Acute alcoholism* (2) *Chronic alcoholism.* (3) *Delirium tremens* (4) *Various other manifestations* (i) Korsakow's syndrome, (ii) 'Wet brain' (iii) Acute hallucinosis, (iv) Alcoholic automatism; (v) Dipsomania, (vi) Relation to other diseases insanity, tuberculosis, pneumonia (5) *Special forms of alcohol poisoning*

The amount constituting excess is greatly dependent on idiosyncrasy. A healthy adult male should not exceed ℥ij *of ethyl alcohol* daily as routine

Hereditary Influence Epilepsy is more prevalent in descendants of alcoholics. Extensive alcoholic excess tends to degeneration in a race usually a race qualified by mental deficiencies for the extinction of the unfit

1. ACUTE ALCOHOLISM

Characterized usually by (i) Flushed appearance and congestion, (ii) Incoordination (iii) Lack of mental control Unconsciousness may follow, in which (a) Person can be roused temporarily; (b) Pupils are dilated (c) Pulse is full (d) Respirations are not stertorous, (e) Temperature is subnormal

DIAGNOSIS From other forms of coma (see COMA, p. 204)

Acute Alcoholic Coma. Occasionally rapid onset following large amounts of undiluted spirits Condition of collapse

Treatment. A debauch involves a long sleep, a purgative, and a day of nausea and disgust

ACUTE ALCOHOLIC COMA Wash out stomach with tube at conclusion pour in a pint of warm sodium bicarbonate solution with magnesium sulphate ℥ij, or hot coffee. Hot bottles, and prevent a chill

ACUTE ALCOHOLIC MANIA Inject apomorphine gr. $\frac{1}{10}$ to $\frac{1}{6}$.

2. CHRONIC ALCOHOLISM

Has effects upon (a) The mental condition and nervous system, (b) The tissues, causing degeneration of cells and fibrosis Results vary somewhat with form of alcohol beer tends to grossness and gastritis spirits to leanness and fibrosis and cirrhosis of the liver. Patient feels worse in morning before mitigation by further alcohol

MENTAL CHANGES. Concentration, memory, and judgement deficient. *Carelessness of clothes.* Irritability and brutality often marked. *Epileptic fits* may occur.

FACIES.—Congested. Venules dilated. Nose large and red may be 'acne rosacea'. Conjunctivæ water often icteroid.

Alcoholism—Chronic, *continued.*

ALIMENTARY SYSTEM.—Tongue furred, breath heavy. Headache. Nausea and lack of appetite, especially in the morning. Constipation. Stomach: (a) Chronic atrophic gastritis, with deficient HCl; (b) Dilatation, common in beer drinkers.

VOICE often husky: or, in gin drinkers, shrill.

TREMOR of hands and tongue.

Other important effects are:—

MULTIPLE PERIPHERAL NEURITIS (q v.).

CIRRHOSIS OF LIVER.—Also fatty cirrhosis of liver: enlarged and tender: especially with malt liquors.

CIRCULATORY SYSTEM.—Heart: fatty degeneration common. Arteriosclerosis.

KIDNEYS.—Chronic nephritis common.

MORBID ANATOMY OF CENTRAL NERVOUS SYSTEM.—Changes slight. In nerve cells: experimentally chromatolysis, disintegration of Nissl's granules; but lesions are *not permanent*, and recover on remitting alcohol. Chronic hæmorrhagic pachymeningitis with adhesion of dura not uncommon.

Tuberculosis and pneumonia frequent, and mortality high.

Treatment—Effective only in an institution. Basis:—

1. COMPLETE WITHDRAWAL OF ALCOHOL. For a few days rapidly diminishing quantities are given.
2. ATROPINE AND STRYCHNINE.—Increasing amounts, up to full doses in about 3 weeks, then diminishing.

SUBSEQUENTLY ENTIRE ABSTENTION. Relapses common.

IN MILD FORMS, a stomachic mixture is useful:—

℞ Pot. Bromidi	gr v	Tinct. Capsici	♏iv
Tinct. Hyoscyami	♏xxx	Aq. Menth. Pip.	ad ℥j
Tinct. Nux. Vom.	♏v		

t.d.s. before meals.

3 DELIRIUM TREMENS.

Occurs in *persistent drinking*, generally under stimulus of temporary unusual amounts, or sudden cessation or shock. Pneumonia and fractures frequently result from alcoholism, and lead to delirium.

ONSET.—Never sudden. Insomnia, depression, and restlessness for few days. May be hallucinations of animals, at this stage *recognized as imaginary*. Also bad dreams.

DELIRIOUS STAGE:—

NOISY DELIRIUM. Loss of orientation of time and place.

HALLUCINATIONS OF SIGHT.—Rats, snakes, loathsome forms. Characterized by: (*a*) Terror ('the horrors'), (*b*) Animals creeping over body, (*c*) Animals numerous.

HALLUCINATIONS OF SOUND not common.

TREMORS.—Especially hands and tongue.

INSOMNIA.

Tongue: thick fur. *Pulse*: rapid. *Temperature*: 100° to 102°.

PROGRESS:—

1. RECOVERY.—Usually following a long sleep. Duration of

delirium two to five days. Subsequent hazy memory of occurrence, but often distinct recollection of hallucinations.

2. INSOMNIA PERSISTS.—Passes into condition of 'wet brain', or prostration and cardiac failure.

MORTALITY.—High with pneumonia: otherwise usually recovery. Occasionally suicide.

Diagnosis.—Simple, but examine for pneumonia and fractures

Treatment.—

GENERAL TREATMENT.—Indications are to procure rest and support the heart. *Confine to bed* in quiet room. Restraint by tactful nursing often necessary, may be accomplished by sheets fastened across ankles and across chest. *Alcohol*: *Withdraw* at once unless patient is old or weak, when withdraw rapidly in thirty-six hours. *Aperient*: Calomel gr. ij to v. If high temperature, cold packs. Emetics weaken heart.

CARDIAC STIMULANTS.—Inject caffeine sod.-sal. gr. ij every eight hours; or strychnine; or ammonia and ether by mouth

DIET. Milk and eggs, every two or three hours (never waken).

SEDATIVES.—

1. Chloral hydrate gr. xxx: may be repeated three times on first day. If by rectum, double dose. Not with cardiac weakness. Inject morphia gr. ¼ to gr. ½ also if necessary.
2. Hyoscine hydrobromide, inject gr. $\frac{1}{100}$ to $\frac{1}{150}$, with morphia gr. ¼ to ½. Especially in severe delirium in young subjects.
3. Apomorphine: inject gr. $\frac{1}{6}$ to ¼. Quiets a truculent subject. Can be combined with hyoscine. Chloral may follow either of these.
4. Potassium bromide gr. xl: inferior to chloral hydrate.

IN CONVALESCENCE. Hyoscyamus and capsicum mixture as in CHRONIC ALCOHOLISM, p. 290.

4. VARIOUS MANIFESTATIONS.

Korsakow's Syndrome or Psychosis.—

Only in persistent alcoholism, usually in middle-aged 'tipplers'. Not uncommon.

ONSET of mental condition may be: (*a*) Gradual; (*b*) Sudden; (*c*) Following mild delirium tremens.

SYMPTOMS.—Two factors:—

(1) MULTIPLE PERIPHERAL NEURITIS.—May precede or follow the onset of mental disturbances.

(2) PECULIAR DEFECTS OF MEMORY.—(*a*) Loss of orientation of time and place, e.g., last week's event put years ago, environment outside the room door forgotten; (*b*) Loss of memory of periods, mainly recent, the irregular gaps being filled with complex fabrications. Patient often forgets immediate relatives. Yet intellectual reasoning is often surprisingly correct.

COURSE AND PROGNOSIS.—Very prolonged. If alcohol is withheld, mental improvement continues for months or years, but probably is never complete.

Alcoholism—Various Manifestations, *continued*

TREATMENT.—Symptomatic

4. **'Wet Brain'** (*Alcoholic serous meningitis*) Only in chronic alcoholism, usually following delirium tremens The noisy delirium changes to low and muttering type Pallid Prostrated Lies on back, extending arms and hands towards ceiling Tremor marked No paralysis or optic neuritis May progress to coma with rigidity of muscles and neck

TERMINATION (1) Death from cardiac failure or pneumonia, mortality at least 50 per cent (2) Slow recovery

MORBID ANATOMY Serous fluid in meninges a transudate and not true meningitis

TREATMENT Feed regularly milk and eggs Inject aqueous sod salicyl gr ij eight hourly or strychnine gr ¼ six hourly

5. **Acute Hallucinosis.** Auditory hallucinations and ideas of persecution delirium slight Suicide frequent Forms merge into delirium tremens

6. **Alcoholic Automatism.**—May follow even a mild debauch with previous alcoholism head injuries, epilepsy or sunstroke Automatism for various periods journeys or business often performed suddenly wakes up days later oblivious of interval

7. **Dipsomania.**—Periodic impulse for an alcoholic debauch Causes various may be a recurring psychosis or depression with increasing craving until irresistible Intervals tend to shorten and chronic alcoholism tends to develop

TREATMENT Control if possible during period of onset

8. **Relation to Insanity, etc.** Chronic alcoholism may lead into dementia but alcoholism accounts for only small proportion of insanity

5. SPECIAL FORMS OF ALCOHOL POISONING

Methyl Alcohol (*Wood alcohol*)—Used to adulterate cheap spirits etc Several outbreaks recorded e.g. Berlin 1911

CHARACTERISTICS

1. ONSET OF SYMPTOMS *Delayed* for twenty four to seventy two hours
2. BLINDNESS Few severe cases escape Stages are (a) Bilateral total blindness retrobulbar neuritis few hours or days after intoxication (b) Partial recovery (c) Permanent blindness develops with optic atrophy days or weeks later In milder types central scotomata contraction of visual fields
3. UNCONSCIOUSNESS Frequently passing into coma Coma practically always fatal

Ordinary symptoms of intoxication also occur

TREATMENT —Stomach wash, within twelve hours of ingestion Sodium bicarbonate ʒj two hourly by mouth for six doses, or intravenously,

Absinthe.—Acute or chronic excess causes convulsions Chronic excess causes neuritis, hyperæsthesia, and hallucinations also

CHAPTER L.

OPIUM POISONING. MORPHIA HABIT.

Acute Opium Poisoning.—Usually from suicidal intention.

SYMPTOMS.

COMA. Profound; onset gradual.

PUPILS. Contracted; unequal (may dilate in final stage).

RESPIRATION. Slow; cyanosis.

Skin moist. Temperature normal. Plantar reflex flexor.

TREATMENT. (Acute effect of opium is mainly on the respiratory centre. Morphia, even when injected, is excreted into the stomach and reabsorbed.)

INDICATIONS. (1) Wash out stomach hourly with pot. permanganate 1 1000, leaving a few ounces each time. (2) Artificial respiration when and as long as necessary. Enemata of hot coffee. Old method of keeping subject awake by slapping, etc., combined with large injections of atropine, now [illegible]

Morphia Habit.—Usually commenced for pain, especially for continued lingering types. Generally the dose has to be increased gradually. Several months elapse before symptoms commence.

APPEARANCE. Prematurely aged; sallow, emaciated; hair thin and gray.

PUPILS. Dilated or unequal except under a dose.

UNRELIABILITY AND LAXITY. Character [illegible]; [illegible] deficient; irritable. Nervous [illegible] symptoms. May be at first. Digestion and nutrition impaired.

ON WITHDRAWAL OF DRUG. [illegible] Lassitude and mental depression. Nausea, vomiting, and abdominal pains. *Dread of death is present.* Suffering is great, and craving for drug so intense that subject will adopt any conceivable means to obtain it. Insomnia and nocturnal hallucinations often, always terrifying, most commonly of sight. Restlessness. Pulse and respiration slow. Constipation extreme. Sensory disturbances common, as hyperæsthesia of feet. Gradually, in absence of drug, disturbances subside and convalescence commences.

Treatment.—In morphia habit, drug no longer gives pleasure, and most subjects desire but dread, to stop it. The sufferings are so intense that practically one may say *none can withstand it, except in absence of regular treatment in a home or institution.* Original cause frequently a painful condition: ascertain and treat it if possible before commencing cure, otherwise pain will return and relapse is almost certain.

METHODS OF TREATMENT.—Without other drugs, sudden withdrawal unjustifiable, from frequency of fatal collapse: even with gradual withdrawal, sufferings intense. Most satisfactory

Morphia Habit—Treatment, *continued*

method depends on large doses of other drugs: fatal collapse also avoided.

HYOSCINE METHOD.—Inject hyoscine hydrobromide gr. $\frac{1}{100}$ and subsequently gr. $\frac{1}{150}$ hourly until mild delirium and dilated pupils: then every two to three hours up to forty-eight hours to maintain delirium. No morphia given. On recovery of consciousness, has lost craving for drug. Subsequently, inject pilocarpine gr. $\frac{1}{4}$ and repeat several times; this causes sweating and aids elimination of hyoscine.

In other similar methods, morphia is more gradually replaced by atropine, hyoscine, or hyoscyamus.

Subsequently there may be diarrhœa, or pains in joints.

AFTER-TREATMENT.—General health is regained with surprising rapidity, but must be aided by exercises, full diet, and good air. Appetite may be excessive. Bowels need regulation. Treatment and observation should continue for months, with every precaution against obtaining drug.

CHAPTER II

COMA.

Methods of Investigation.—

A. GENERAL HISTORY AND DATA.—Obtained from friends and others.

PREVIOUS HISTORY.—(i) Existence of any form of disease, e.g., renal, diabetic, nervous, or epileptic. (ii) Previous similar attacks. (iii) Alcoholism. (iv) Prodromata, e.g., headache, giddiness, vomiting or convulsions. (v) Drugs found with patient.

ONSET OF COMA.—(i) Injury. (ii) Alcohol. (iii) Rapidity of onset, convulsions, and general account.

B. EXAMINATION OF PATIENT.—

APPEARANCE.—Congestion, cyanosis, respiration, depth, rate, stertor, blood on lips.

EXAMINE HEAD FOR INJURY.—Skull for fracture, ears and nose for blood or meningeal fluid, subconjunctival hæmorrhages.

PARALYSIS.—Especially *unilateral*. Note: (i) The cheeks puffed out on paralyzed side. (ii) Flaccidity of paralyzed limbs, drop 'dead'. (iii) Conjugate deviation of eyes and head. (iv) Reflexes, tendon and abdominal, absent on paralyzed side, and Babinski's sign present.

PUPILS.—Size, equality, reaction to light.

BREATH.—Alcohol, acetone.

HEART.—Presence of murmurs, etc. *Condition of pulse and*

TEMPERATURE.
URINE Sugar, albumin.
Special examinations —
FUNDI —Albuminuric retinitis, optic neuritis
BLOOD PRESSURE.
LUMBAR PUNCTURE.—If meningitis suspected
GASTRIC CONTENTS. —To be preserved if poisoning suspected

—The most important may be given in five groups —
ALCOHOL.
EPILEPSY.
INJURY AND DISEASES OF THE HEAD *Cerebral hæmorrhage* and various nervous lesions meningitis, encephalitis, intracranial tumour, abscess or embolus (rarely), sinus thrombosis Rarely dementia paralytica
OPIUM Also other narcotics chloroform, chloral, bromide, veronal, carbolic acid, oxalic acid, carbon monoxide
URÆMIA, ACIDOSIS, and CHOLÆMIA —Nephritis diabetes, eclampsia, hepatic diseases, acute yellow atrophy, etc

Other causes —
MALIGNANT MALARIA
SEVERE FEVERS Later stages Enteric, typhus, dysentery, cholera Yellow fever (cholæmia) Blackwater fever
SEVERE HÆMORRHAGE Internal or external Gastric duodenal, enteric, ectopic gestation, etc
HEAT STROKE Also extreme cold
HYSTERIA
LEAD ENCEPHALOPATHY ENCEPHALITIS LETHARGICA

Differential Diagnosis —

ALCOHOL.—Coma rarely complete Respiration deep but not stertorous Pupils dilated Temperature subnormal. Alcoholic breath

EPILEPSY Post epileptic coma Coma short about one hour History of previous attacks or signs of a fit, e.g., tongue bitten

CEREBRAL HÆMORRHAGE —Onset sudden Coma deep Unilateral paralysis (*see also e*) Pupils dilated may be unequal (larger on affected side) Temperature normal. Note age, condition of arteries and heart, fundi For localisation of site, *see* CEREBRAL HÆMORRHAGE *Pontine hæmorrhage*: paralysis bilateral, pupils contracted, pyrexia, may be crossed paralysis *Meningeal hæmorrhage*: history of injury, latent period, gradual onset of coma

OPIUM AND MORPHIA Onset of coma gradual *Pupils pinpoint Respiration slow.* Skin moist Temperature normal. Plantar reflex flexor

NARCOTIC DRUGS Pupils dilated Analysis of stomach contents, drugs, etc In carbon monoxide poisoning, peculiar cherry red appearance

URÆMIA —Onset of coma gradual. Pupils contracted. Respiration may be Cheyne Stokes type. Convulsions common Note urine, fundi, blood pressure, arteries May be œdema. Prodromata usual: headache, vomiting, and convulsions.

Coma—Differential Diagnosis, *continued.*

DIABETES.—Onset of coma gradual. Air-hunger. Acetone in breath. Glycosuria, ketonuria. Prodromata usual: headache, anorexia, epigastric pain.

HYSTERIA.—Pulse and respiration normal. No cyanosis. Resists lifting of lids.

CEREBRAL EMBOLUS (rare cause).—Cardiac disease.

SEVERE HÆMORRHAGE.—Extreme blanching. Pulse very rapid.

MALARIA AND HEAT-STROKE.—Hyperpyrexia. In malaria, protozoa in blood.

CONCUSSION.—Pupils unequal. Other signs slight. (Concussion to be diagnosed with great caution during coma.)

Diagnosis in other conditions depends mainly on associated diseases, on prolonged observation, and other factors.

Notes.—

PARALYSIS PRESENT. Cause is cerebral hæmorrhage, thrombosis, or embolism.

ALCOHOL, INJURY, AND HÆMORRHAGE. Often co-exist: diagnosis difficult and catastrophes common. *Treat all doubtful cases as serious.* Alcohol in breath is no guide.

INTERNAL HYDROCEPHALUS. A possible cause of rare cases of prolonged coma.

CHAPTER LII

LEAD POISONING.

Lead poisoning may be acute or chronic, usually the latter. It arises from many causes, the most important being (1) *Industrial*; (2) *Accidental*; (3) *Medicinal*; (4) *Adulterations*.

Industrial.—Lead miners, in carbonate mines only (Broken Hill); rare in metallic lead mines. Smelters, workers in white lead factories, painters, plumbers, enamel plate makers, glazers and potters. File makers.

Accidental.—

WATER, if slightly acid, dissolves lead rapidly, especially from new pipes. Old lead surfaces often have covering of lead carbonate, partially protective, but soluble in hot water. Water from peaty soil dangerous from presence of humic acid, which dissolves lead.

CIDER AND BEER, in contact with lead. The first morning drink from a bar may contain lead; drunk daily by a barman can produce poisoning.

Medicinal.—Rare, except diachylon taken as abortifacient.

Adulterations.—Lead chromate has been used as colouring in baking powder.

Dose.—Doubtful. Brouardel states that one milligramme ($\frac{1}{70}$ gr.) daily produces plumbism.

Path of Entry. (1) *Intestinal tract*, from lead on hands or swallowed with saliva, etc.; (2) *Respiratory tract*, inhaled as dust; (3) *Skin*, practical importance slight.

Paths of Excretion. Urine and fæces. Recognition simple by electrolytic method.

Etiology.—*Age*. Liability is greater in youth. *Sex*: Females are especially susceptible. Idiosyncrasy is marked (Oliver). Excess of alcohol predisposes.

Morbid Anatomy.

ACUTE FORM. Changes as in gastro-enteritis.

CHRONIC FORM. Chronic catarrh of stomach and intestines. Cæcum and ascending colon may be dusky from lead deposited in mucous membrane (well seen in microscopic sections). Liver contains much lead. Peripheral nerves show degeneration. Interstitial nephritis common.

CEREBRAL FORMS. Œdema of brain and minute hæmorrhages.

ACUTE LEAD POISONING.

Rare; practically confined to large doses of lead acetate (sugar of lead). Vomiting and abdominal pain and symptoms of gastro-intestinal irritation. May be fatal.

CHRONIC LEAD POISONING.

Effects of chronic lead poisoning are: (1) Certain general symptoms. (2) Three classical chronic types, occurring in order of frequency, as: (a) Colic. (b) Paralysis. (c) Encephalopathy. (d) Certain remote effects.

A. General Symptoms.

ANÆMIA AND PALLOR.

CONSTIPATION. General lassitude, nausea and disturbed digestion, headache.

BLUE LINE ON GUMS. Near but not at margin of gums. Usually lower jaw. Due to H$_2$S given off forming insoluble black sulphide; hence commoner with carious teeth, and may be absent with good teeth. In deep layers of mucous membrane and not removable by brushing; in papillæ hence line discontinuous under lens. May appear within a week of exposure. An external removable deposit may be present at margin. Duration at least three weeks after cessation of exposure.

BLOOD CHANGES. Saturnine cachexia: Hæmoglobin and red cells diminished (2,000,000 to 3,000,000 per c.mm.). *Basophilic degeneration*, stippling of red cells common and often marked, but not proof of lead poisoning. Normoblasts relatively numerous. Leucocytes, little change.

ABORTION. Very common. Menstruation irregular.

Lead Poisoning—Chronic, *continued*

B. **Classical Clinical Types.**—Usually preceded by general symptoms.

(1) COLIC (Lead or painters' colic) - The most common type Pain paroxysmal, eased by pressure General distribution, occasionally unilateral or localized Pyrexia rare Pulse slow, often high tension and small Constipation usually obstinate Urine reduced Duration three to ten days: recurs with further lead Never fatal Diagnosis from appendicitis by apyrexia and slow pulse

(2) LEAD PALSY —Onset subacute Occasionally previous tinglings, tender calves etc, but usually not With paralysis rapid muscular atrophy sensation normal tremor common, reaction of degeneration develops May be pains in joints Types of paralysis —

WRIST-DROP —Paralysis of extensors of wrist and fingers Bilateral *Supinator longus escapes* also extensor ossis metacarpi pollicis all others supplied by musculospiral nerve affected Extensor longus pollicis affected first, inability to extend terminal phalanges When severe prominence on back of wrist (Grubler's tumour) Is commonest type

When following occur are usually subsequent to above

BRACHIAL TYPE Duchenne Erb or scapulo humeral type. Uncommon Deltoid biceps brachialis anticus, and supinator longus Deltoid earliest and most severe arm hangs to side *Note* When this type co exists with wrist drop supinator longus is thus affected

RARE FORMS (a) Aran Duchenne type Small muscles of hand viz, interosseii, thenar and hypothenar eminences Atrophy marked May precede wrist drop (b) Peroneal type 'Toe drop' Mainly in children Tibialis anticus escapes

GENERALIZED PARALYSIS Very rare Usually commences as wrist drop, and extends Very rarely onset acute resembling Landry's paralysis or acute febrile polyneuritis

CRANIAL NERVES Practically never affected (except rarely optic neuritis occurs)

Prognosis Depends on extent and duration of paralysis With treatment and removal from lead, recovery is good in early cases

(3) LEAD ENCEPHALOPATHY Rare, but high mortality Onset acute, but usually previous symptoms of plumbism

SYMPTOMS may be (I) *Convulsions*, identical with epilepsy (II) *Acute mania*, violent, (III) Delirium, (IV) Coma *Optic neuritis* and atrophy may occur. Permanent insanity rare

Rarely, symptoms resemble dementia paralytica, but are curable.

C. **Remote Effects.**—

ARTERIOSCLEROSIS and ancillary conditions, e g, chronic nephritis and myocarditis, are common in those long exposed to effects of lead

GOUT — Association formerly over-emphasized

Diagnosis of Chronic Poisoning.—Principal characteristics (1) Anæmia and cachexia; (2) Constipation, (3) Blue line, (4) Colic, (5) Wrist drop (supinator longus escapes) Lead present in urine and fæces (by electrolysis, etc)

Treatment. Remove from exposure to lead. Treat general health

COLIC AND CONSTIPATION Relieve pain by warmth or hot bath if severe inject morphia gr , and atropine gr , Open bowels with olive oil enemata continue with mag sulph ℥j, tinct belladonnæ ♏x, t d s or oftener, in order to obtain free motions

When bowels are open, give pot. iod. gr. v, t d s. (Action much discussed KI supposed to aid elimination of lead in a soluble form, and thus may cause acute symptoms Others deny any effect)

LEAD PARALYSIS Treat as other paralyses protect muscles by splints from overstretching, massage, exercises electricity

ENCEPHALOPATHY Sedatives bromides or hyoscine Hot packs In severe forms, venesection or lumbar puncture

CONVALESCENCE Treat anæmia with arsenic and tonics (iron tends to constipate) *Recurrences* occur frequently after an initial attack if exposure continues

Prophylaxis.—

FACTORIES Many laws in force for ventilation, cleansing, preventing dust, etc

WORKMEN *Cleanliness* wash hands before meals weekly bath Meal before work (protein hinders absorption of lead) Milk to drink Sulphuric acid lemonade.

CHAPTER LIII

ARSENIC POISONING.

Opportunities for arsenic poisoning occur in many circumstances. (1) Mining and smelting, (2) In various arts for colouring purposes e g, wall papers, carpets, artificial flowers, (3) In tanning hat making, and various processes connected with skins, (4) Medicinal, (5) For poisoning animal life 'Rough on Rats', fly papers *Industrial* arsenic poisoning is not very common

IN MINES — A fatal chronic bronchitis occurs also found in cobalt and other mineral mines containing arsenic often known locally as 'cancer of the lungs'.

Arsenic Poisoning, *continued.*

COLOUR-MAKERS.—Use of arsenic controlled by law. Produces both green and bright-red colours, e.g., Persian red.

WALL PAPERS, ETC.—Certain moulds (*Penicillium brevicaule, Mucor mucedo*) produce a volatile organic arsenic compound

MEDICINAL.—Widely employed as tonic for anæmia Children bear it well. Idiosyncrasy is marked. Maidens have taken it internally to obtain 'peach-blossom' complexions, and strong men to ward off fatigue (Styria): the latter use suggests possibility of *tolerance* being established.

Elimination.—By all excretions and secretions, urine, fæces, milk. Present in hair and various tissues Elimination is rapid

Chemical tests (Marsh and modifications) are of great delicacy. Before it was realized that traces might be present in a normal body, this delicacy led to judicial errors in murder trials, e.g, Marie Lafarge, Darval

Action of Arsenic. Toxic to all protoplasm, e g, spirochætes, man In small doses, widely beneficial: nutrition, blood, skin digestion, nerves. In blood, especially increases number of red cells in anæmia.

Morbid Anatomy.—

ACUTE POISONING. - Gastro enteritis stomach, duodenum, colon, and rectum; may be some ulceration, but not perforation Fatty degeneration of liver, etc, absent or slight Exhumed bodies show remarkable preservation.

CHRONIC POISONING. —Rarely fatal Degeneration of peripheral nerves: atrophy of anterior horn cells

Forms of Poisoning.—Mainly as, (1) *Acute*, murder, suicide, accidental. (2) *Chronic*, especially medicinal (3) Various occasional forms tending to possess dramatic features, e g, beer epidemic, atoxyl, chimney-sweeps' cancer, ferrosilicon, salvarsan

1. ACUTE ARSENIC POISONING.

Characterized by gastro-enteritis.

ONSET.—Interval of ¼ to ½ hour after ingestion rarely, a few hours.

SYMPTOMS.—

Pain in stomach: burning, agonizing: spreads over abdomen *Vomiting* and then *diarrhœa*, violent and repeated *Thirst* extreme. Temperature low. Pulse feeble Restlessness Occasionally metallic taste in mouth (rarely garlicky). Constriction in throat. Cramps in calves, severe, but often absent.

PROGRESS —Collapse and rapid death Or symptoms remit and return, with death in twenty-four to forty eight hours or rarely several days.

General resemblance to cholera', may be profuse *watery stools*

TREATMENT.—Stomach tube. Mild emetic if no vomiting

(mustard ℥ss to a tumbler). Milk to drink. Freshly prepared ferric hydrate (tinct. ferri perchlor. ℥j in a glass of water; add magnesia, washing soda, or *dilute* ammonia ℥j, strain off precipitate in handkerchief and dissolve it in a glass of hot water, give repeatedly).

MINIMUM FATAL DOSE: Gr. ij.

2. CHRONIC ARSENIC POISONING.

Effects especially *skin, mucous membranes, and nerves.* May follow recovery from a single large dose. Frequently follows medicinal treatment, owing to large doses being beneficial in many conditions.

MILD EARLY SYMPTOMS IN MEDICINAL TREATMENT:
(1) *Headache*, earliest. (2) *Conjunctivitis and watery eyes.* (3) *Silvery tongue* (may be absent). (4) *Nausea.* More advanced: flushings, nasal and respiratory catarrh, tingling of extremities.

CHRONIC SYMPTOMS: Four groups (Taylor).
- GASTRIC SYMPTOMS: Nausea, vomiting, diarrhœa. Nutrition impaired.
- CATARRH OF MUCOUS MEMBRANES: *a) Eyes*, conjunctivitis and chemosis, puffy eyelids. *b) Nose and mouth*, rarely perforation of septum. *c) Lungs*, bronchitis.
- SKIN LESIONS.
- PERIPHERAL NEURITIS.

Last two groups form the special and diagnostic characteristics.

SKIN LESIONS:
- PIGMENTATION: *Early and common.* Affects areas exposed, subject to pressure, or previously pigmented. Yellow to deep brown. Buccal mucous membrane escapes: *compare* ADDISON'S DISEASE. Fades partially on treatment, but not completely if severe.
- KERATOSIS: Soles and palms. Of all degrees, desquamation marked. 'Corns' may form. Occasionally gives rise to epithelioma.
- HERPES: Common. Is produced by no other drug.
- ERUPTIONS: Of numerous types: bullous, psoriasiform, etc.
- NAILS become brittle. HAIR falls out.
- VASOMOTOR PHENOMENA: Occasionally resembling erythromelalgia.

PERIPHERAL NEURITIS.—Both sensory and motor fibres, especially lower extremities.
- SENSATION: *Pain marked.* Calves tender. Later, sensation lost.
- PARALYSIS: Lower extremities first, *especially toes.* All muscles, but extensors more than flexors. Atrophy rapid. Reaction of degeneration present. Knee jerks absent. Deformities may follow.
- Arms later and less often affected.

MENTAL SYMPTOMS rare.

PROGNOSIS. Improvement rapid unless condition very severe.

Arsenic Poisoning, *continued.*

3. OCCASIONAL FORMS.

MANCHESTER BEER EPIDEMIC.—Arsenic was suggested by frequency of herpes (Reynolds). Arsenic was traced to glucose used in brewing; origin being from sulphuric acid prepared from pyrites containing much arsenic.

ATOXYL.—Often produces rapid optic atrophy within seven to ten days of injection.

CHIMNEY-SWEEPS' CANCER OF SCROTUM. Ascribed to arsenic in soot (doubtful).

FERROSILICON. When moist, gives off AsH_3 and PH_3. Many deaths occurred in ships and barges before recognition. Very toxic, causing collapse and death in a few hours.

ARSENOBENZOL PREPARATIONS.—Very rare. (*See* p. 275.)

Diagnosis.—In cases of slight general ill health, cardiac weakness, etc., diagnosis only by finding arsenic in excreta, hair, or suspected articles.

PIGMENTATION IN ADDISON'S DISEASE. Buccal mucous membrane often affected. (*See* Pigmentation of Skin, p. 301.)

LEAD PALSY. Affects upper extremities; certain muscles escape; sensation normal. No local pain. Special symptoms of lead poisoning present.

ALCOHOLIC NEURITIS. Usually delirium; affects calves rather than toes. Often difficult. (*See* MULTIPLE NEURITIS.)

CHAPTER XIV.

VERONAL POISONING.*

Veronal is diethyl barbituric acid, officially (B.P.) known as *barbitonum*. Only slightly soluble in cold water; more so in hot water. Bitter taste; unpleasant in milk. Powerful hypnotic. *Dose*: gr. ½ to x; gr. v sufficient for adult. Many fatalities.

Acute Veronal Poisoning.

SYMPTOMS.—Single excessive dose produces drowsiness, headache, and may be ataxia and reeling gait; nausea unusual. Deep sleep follows, progressing to coma; cyanosis, and rapid often stertorous, respiration. During coma, pyrexia common; may be physical signs suggesting pneumonia (may be erroneously diagnosed). Rashes unusual; hæmaturia doubtful. From deep coma recovery rare.

FATAL DOSE.—In healthy adult, 50 gr. is 'average minimum fatal dose'. Smaller doses often fatal with disease or other drugs.

* *See* especially Willcox, *Lancet*, 1913.

TREATMENT.

1. STOMACH WASH.—Especially valuable within four hours owing to insolubility, but advisable even later. Leave in stomach hot strong coffee, 1 pint with some milk and castor oil ℥j. Retain washings for analysis.
2. CARDIAC STIMULANTS.—Camphor, caffeine, strychnine. Oxygen if cyanosis. Saline may be injected intravenously or per rectum. If retention, catheterize bladder; retain urine for analysis; veronal rapidly excreted by kidneys. Feeding necessary in prolonged coma (stomach tube).

AUTOPSY.—Signs of death from gradual cardiac failure—no characteristics. Cyanosis. Heart dilated, especially right side. Hypostatic congestion of lungs.

Veronal Habit.—Mental and psychical disturbances may develop. General condition suggests chronic alcoholism with tremors, ataxy, and thick speech. Tolerance is slight, and an overdose is often fatal, especially with constipation or renal disease.

CHAPTER IV.

COCAINE POISONING.

Cocaine is an alkaloid in methyl benzoyl ecgonine. Cocaine is in the leaves of *Erythroxylon coca*. It is usually used as a local anæsthetic. In *habitués* other drugs are also taken, which complicate the symptomatology.

Acute Cocaine Poisoning.

DOSAGE.—Injection should not exceed [illegible]. Individual idiosyncrasy very marked.

SYMPTOMS.—

MILD DEGREES.—Faintness, giddiness, rapid pulse and respiration, restlessness, nervous excitement and anxiety, no pleasant sensations.

SEVERER DEGREES.—Nervousness and great restlessness. Pulse rapid and feeble. Pupils dilated. Perspiration, nausea and vomiting. Collapse may occur with or without loss of consciousness. Respirations variable, may be slow, irregular, or Cheyne-Stokes, and with cyanosis. Convulsions may occur, often violent. Occasionally mania, but unconsciousness more frequent. Pulse often slow before death.

AFTER EFFECTS.—Insomnia, giddiness, anæsthesias.

TREATMENT.—Recumbent. Stimulants: alcohol, camphor, or caffeine. Artificial respiration or oxygen for respiratory failure. Strong coffee enema. Wash out stomach if taken by mouth.

Cocaine Poisoning, *continued.*

Cocaine Habit.—Usually by snuffing or hypodermic injection.

EARLY STAGES.—Usually taken intermittently. Pleasant sensations of exhilaration, mental power, and physical strength subject talkative and happy.

LATER STAGES.—Rapid moral and physical degeneration Subject pale and emaciated. Depressed and irritable; when under drug, voluble but disconnected Insomnia. Muscular restlessness, may be irregular choreiform movements. Movements often clumsy. Paraesthesias, especially sensation of small foreign bodies under the skin, often at finger-tips. Mental changes: hallucinations of voices common, delusions of persecution, jealous, sexual, and obscene

TREATMENT Institutional treatment correct remedy. Sudden and complete withdrawal is without danger. The hyoscine method (*see* MORPHIA HABIT, p 294) may be employed

CHAPTER LVI

FOOD POISONING.

Diseases of many kinds may be conveyed by or arise from ingestion of food, but the term 'food poisoning', though not clearly defined is usually applied to certain acute conditions, mainly gastro-enteritis or collapse, generally due to meat or fish, and often occurring in so-called epidemics or outbreaks, attacking a number of persons within a short space of time

1. FOOD POISONING FROM MEAT AND FISH.

Causes. (1) Infection with bacilli, (2) Products of bacillary action (toxins) or of putrefaction (ptomaines).

Modes of Contamination.—

1. BACILLI PRESENT (*a*) Animal infected and sick when slaughtered, (*b*) Food, during preparation for consumption contaminated by human 'carrier' or by flies
2. PRODUCTS OF BACILLARY ACTION (*Ptomaine poisoning*—Selmi, 1878)—Bacilli during growth often produce toxins, which may survive processes killing the bacilli, and later cause illness: probable frequent cause of 'tainted food' poisoning Putrefaction may similarly produce toxins Alteration of appearance and taste occurs only with putrefaction

   'PTOMAINE POISONING'. Formerly believed that by autolysis in meat or fish, or by putrefaction due to bacteria poisonous substances (ptomaines) were formed which caused food poisoning These ptomaines were considered to result from protein disintegration, e g., putrescine and cadaverine. Term now little used: applicable only when no bacilli are found.

Substances Affected.—Pork, veal, and beef most commonly, mutton rare.

Two Principal Groups.—(1) With gastro enteritis, common form. (2) With nervous symptoms, very rare.

GROUP WITH GASTRO-ENTERITIS.*

Commonly associated with *B. aertrycke* and Gaertner's bacillus

Bacteriology.—

(i) Gaertner, 1888, isolated from a food poisoning outbreak the *B. enteritidis* (Gaertner). (ii) Durham and De Nobele, 1898, independently isolated *B. aertrycke* from a similar outbreak. In addition, (iii) Salmon and Theobald Smith, 1885, described *B. suipestifer* in a swine fever outbreak. (iv) Achard and Bensaude, 1896, isolated from a case resembling typhoid the '*bacille paratyphique*', subsequently redescribed by Schottmuller in 1900 and named by him *B. paratyphosus*, and now known as *B. paratyphosus B*.

Relationship of these Bacilli. Morphological and cultural characteristics are all identical. Agglutinations with specific antisera

1	*B. enteritidis* (Gaertner)	Differentiated	
2	*B. suipestifer*	Identical	Identical
	B. aertrycke		
3	*B. paratyphosus B*		Differentiated

B. aertrycke and *B. suipestifer* are thus identical. The three bacilli are sometimes referred to as the 'Salmonella' or 'food poisoning group', but *B. paratyphosus B* is not considered except by certain German authorities to cause outbreaks of acute gastro enteritis, and its inclusion in a group with the other bacilli is only justified on the basis of its morphological and cultural characteristics; further, its clinical symptoms are practically identical with *B. paratyphosus A*.

Outbreaks of Food-poisoning with acute gastro-enteritis are thus caused by (i) *B. aertrycke*, most often isolated; (ii) *B. enteritidis* of Gaertner. When neither is isolated, with correct bacteriological methods, the cause may be (iii) Toxins of these bacilli, though bacilli are no longer living; (iv) Putrefaction due to *B. proteus* or possibly *B. coli* (doubtful).

The infected food may be normal in appearance, taste, and smell.

Morbid Anatomy. Acute gastro enteritis. Peyer's patches unaffected, no ulceration. Bacilli often recoverable from bile and spleen. In non-fatal cases, affection is mainly of small intestine.

Symptoms. Outbreak of food poisoning usually possesses following features. (1) Symptoms commence almost simultaneously

* *Report on an Epidemic due to B. aertrycke*, Medical Research Committee, Special Reports, 129, Perry and Lidy. *See also* Trevan Fawcett, 'Identification of Enteric, Dysentery, and Food poisoning Bacilli', p. 12.

Food Poisoning—Symptoms, *continued.*

amongst a number of those consuming the food; (2) Illness limited to those eating the food, but *not all necessarily become ill*. (3) In large outbreaks, every degree of severity is usually present. In bacillary forms, excreta of patients are infective, and condition may spread as an epidemic, e.g., in institutions and camps.

LATENT PERIOD.—Variable, three to thirty hours.

ONSET.—Sudden. Abdominal pain and tenesmus, diarrhœa, nausea and usually vomiting. Commonly, headache, cold sweats, often shivering, and syncope when severe.

PROGRESS.—Initial symptoms usually the severest. Diarrhœa often continuous for few hours; rarely severe more than two to five days. Improvement usually rapid. Continued vomiting is most serious symptom, and present in most fatal cases.

PHYSICAL SIGNS.—No characteristic. Tongue clean or slight fur. Abdomen tender but usually not rigid. Spleen not enlarged. No rash. Temperature: in severe cases often 90° to 102°, but may be apyrexial. Character of stools: blood and mucus rare, mucus never in masses as in dysentery. Blood occasionally while motions very frequent.

Sequelæ.—Regulation of bowels difficult, either obstinate constipation or recurrences of diarrhœa. Occasionally, appendicitis.

Mortality.—Low, 1 to 3 per cent. Vomiting usually persistent in fatal cases.

Diagnosis.—Numerous simultaneous cases in household or assembly of individuals. Diagnosis from (1) *Dysentery* by absence of mucus from stools and by specific organisms. *Enteric fever*: by sudden onset and rapid maximum severity.

SPECIFIC DIAGNOSIS.—Bacteriological examination of stools. Serum tests with recognized strains. In an outbreak of any extent, many cases may give negative results, but a few positive examinations are sufficient to establish the cause.

Treatment.—First essentials are *warmth and fluids*, the latter by mouth or by intravenous saline.

EARLY STAGE.—Give castor oil ʒss to ʒj. *For collapse* stimulants.

DIET.—For twenty-four hours fluids only. As diarrhœa ceases diet can be rapidly increased.

DRUGS.—Bismuth 100 to 150 gr. daily. Avoid morphia injections.

℞ Bismuthi Oxycarb. gr. 20 to 25. Aq. Chloroformi ad ℥j.
Tinct. Chloroformi et
Morphinæ Co. ♏5 to 10.
Two- to four-hourly.

Bismuth salicylate gr. 10 to 15 is also a valuable remedy.

VOMITING.—If excessive, wash out stomach; give champagne, epigastric fomentation.

CONSTIPATION subsequently.—Liquid paraffin, ʒij to iv, t.d.s., assisted by enemata.

Investigation of an Outbreak. Note: (1) Clinical symptoms (2) Bacteriological examinations of excreta and serum reactions (3) Epidemiology (*a*) date, time, and number of persons attacked, (*b*) relations to any common meal, or consumption of same article of food, or food prepared by same person or persons (4) Examination of residue of food consumed, especially bacteriologically (5) Mode of preparation of food, cleanliness of kitchen, cooking, and apparatus employed (6) Examination of cooks (*a*) previous or present attack of diarrhœa, (*b*) bacteriological examination of excreta and serum tests, for identification of 'carriers'.

GROUP WITH NERVOUS SYMPTOMS.

Botulism ('*Sausage poisoning*')

Very rare. Usually from uncooked ham or sausages. *B. botulinus* (anaerobe) was isolated by van Ermengem in 1895.

Latent Period.—Twenty-four to thirty-six hours.

Symptoms.—(1) Paralysis of ocular nerves, varies from diplopia, ptosis and dilated pupils to complete ocular paralysis. (2) Thirst, dryness and redness of mouth and fauces, often aphonia. (3) Nausea but no diarrhœa. General muscular weakness. In fatal cases, cardiac and respiratory weakness.

Treatment. Stimulants. Wash out stomach. An antiserum has been prepared, but is generally unobtainable.

2. SHELL-FISH POISONING.

Idiosyncrasy marked in all varieties, children particularly liable.

Mussel Poisoning.—

CAUSE. *Mytilotoxin*, a ptomaine produced by bacteria, isolated by Brieger. Not destroyed by heat and poisonous after cooking.

SYMPTOMS. Onset very rapid, often ten to fifteen minutes after ingestion. Acute collapse, giddiness, coldness and heavy, rapid feeble pulse. No gastro-enteritis. Itching intolerable, either at onset or later. Urticaria common, twenty-four to forty-eight hours. Duration short, but death may occur in few hours. Less commonly, symptoms of acute gastro-enteritis.

TREATMENT. Bed, warmth, and stimulants freely.

Pass *stomach tube* and wash repeatedly with large quantities of water, finally leave in stomach castor oil ℥ij. (Emetics inferior to stomach tube.)

Convalescence rapid, one to two days, but some weakness remains.

Crabs, Lobsters. Idiosyncrasy marked. Gastro-enteritis common; collapse rare.

Oysters. Disease from presence of enteric, Gaertner's bacillus, or both. Oysters 'spoil' readily, producing gastro-enteritis.

Food Poisoning, *continued.*

3. MUSHROOM POISONING.

Idiosyncrasy not uncommon, even to 'edible' varieties.

SYMPTOMS.—(1) Restlessness or actual delirium; (2) Dilatation of pupils and disturbance of vision; (3) Slow pulse; (4) Diarrhœa and vomiting. Symptoms are akin to poisoning by muscarine (which occurs in many mushrooms), but are rarely all present.

SPECIAL TREATMENT.—Wash stomach repeatedly (fungi adhere to wall). Inject atropine sulphate gr. $\frac{1}{50}$: repeat in half hour if necessary (antidote to muscarine).

4. GRAIN POISONING.

Ergotism.—Due to meal made from rye on which ergot fungus (*Claviceps purpurea*) has grown. Chronic condition. Two clinical types, formerly attributed to sphacelinic acid and cornutin respectively:—

GANGRENOUS OR TROPHIC TYPE. Usually toes or fingers. Preceded by tingling, pain, and anæsthesia.

CONVULSIVE TYPE. Preliminary tingling. Then spasms, with flexed arms and extended legs; duration hours or days. Death may occur in convulsions. If chronic, dementia may develop or posterior sclerosis as in tabes.

Lathyrism. Due to certain vetches when powdered (chick pea) being added to cereals. Ascribed to a toxalbumose, comparable with ricin and abrin.

SYMPTOMS.—Onset with sudden severe lumbar pains. Later girdle pains; progresses to spastic or ataxic paraplegia.

Section IV.—DISEASES OF METABOLISM AND DISEASES OF DEFICIENCY.

A DISEASES OF METABOLISM.

CHAPTER LVII

GOUT.

(*P tagra*)

A disorder of metabolism of purin bodies resulting in an excess of uric acid salts in the blood, and characterised typically by attacks of arthritis associated with the deposition of sodium biurate crystals

Etiology.

AGE Most common at between 35 and 50 years, rare before 30.
SEX Males predominate
HEREDITY [illegible] factor
ENVIRONMENT Especially among richer classes
PREDISPOSING FACTORS [illegible] Marked association, especially fermented liquors [illegible] with spirits No great frequency in drunkards with cirrhosis of the liver Beer is factor in poor [illegible] constant excess in rich diet of [illegible] in purins Middle aged and [illegible] living often accustomed to exercise in youth but leading more sedentary in middle life form a large proportion Lead Association formerly noted in South England [illegible] elsewhere action possibly through [illegible]
EXCITING CAUSES OF ATTACK Often forgotten May follow [illegible] or drink, mental worry local trauma, cold
RELATION TO OTHER DISEASES
- GLYCOSURIA Common in late rheum stages importance secondary Diabetic symptoms rare Is an 'alimentary glycosuria'
- URINARY CALCULI Gravel and stone may occur with gout, but no close association
- CHRONIC INTERSTITIAL NEPHRITIS Rarely absent in later stages.

Morbid Anatomy.—

JOINTS. Deposits of acicular crystals of sodium biurate *Earliest site articular cartilages*, immediately below surface If dissolved out by water, cartilage remaining is but little changed In later stages, peri-articular deposits in ligaments, tendon sheaths etc, with erosion of cartilage and deformity of joints Synovial fluid may be turbid with crystals *In acute attack*, signs of hyperæmia, inflammation and joint effusion
TOPHI Deposits of sodium biurate in other tissues, especially where circulation is stagnant or near fibrous tissue: peri-articular or

Gout—Morbid Anatomy, *continued.*

helix of ear, most commonly. Deposits may be scattered throughout the body.

KIDNEYS.—Rarely normal. Changes are: (1) *Chronic interstitial nephritis*: small pale kidney ('gouty kidney'). Less commonly, large red arteriosclerotic. (2) Deposits of urates, intertubular, irregular, or in streaks in pyramids: visible microscopically.

CIRCULATORY SYSTEM.—*Arteriosclerosis*, with ancillary changes of myocarditis and hypertrophy, very common.

Chemical Pathology.—*See* Chapter LVIII, p. 314.

SYMPTOMS.

Clinical manifestations are considered under (1) Acute, (2) Chronic, (3) Irregular, (4) Metastatic gout, (5) Complications.

Acute Gout.—

PREMONITORY SYMPTOMS.—Unusual in first attack, previous health being usually good.

ONSET.—Sudden (especially earlier attacks). In *early morning*.

PAIN.—Intense, as if 'seized in a vice'.

JOINT.—*Swollen, shiny, red, and tender*. Veins near distended. (Later, may pit and desquamate; in larger joints effusion.)

PROGRESS.—During succeeding day, general malaise and irritability, but pain easier. Temperature 100° to 103°. Pain returns at night. Attack lasts about a week; pain gradually lessens, while swelling often increases. Other joints may become affected, prolonging attack to two or three weeks.

JOINTS AFFECTED.—*Great toe* commonest, at proximal joint. Tarsus, ankles, knees, fingers, especially thumb and wrist. Uncommon: elbows, shoulders, hips. Very rare: jaw, sternoclavicular joint.

URINE.—Scanty. High colour. Often trace of albumin and few casts. Deposit of urates and uric acid variable.

Health good following attack. Recovery from first attack complete. Interval between earlier attacks often many years.

Chronic Gout.—

PROGRESS OF DISEASE.—Acute attacks become more frequent. Intervals irregular but shortening: one or two yearly, spring and autumn. Often returns in original joint, then subsequently other joints, or simultaneously several joints affected. Temperature often normal. Attacks of gout may diminish as deposits grow, and often cease in later years.

BETWEEN ATTACKS.—Some pain persists.

JOINTS.—Become deformed, and creak.

PREMONITORY SYMPTOMS.—Common, e.g.: (a) Gastric flatulence and acidity, (b) 'Pricking' in joints, (c) Irritability of temper.

'TOPHI'.—*Chalk stones*. 'Tophaceous gout': (1) Periarticular. Form masses. Skin may slowly ulcerate and expose deposit. (2) Abarticular. Especially in helix of ear. Less common sites: extensor surface of forearm, sclerotics, etc.

URINE. Depends on renal condition. (For uric acid excretion, *see* CHEMICAL PATHOLOGY, p. 314.)

Irregular Gout (*Suppressed gout or 'gouty diathesis'*).—Various symptoms which occur between attacks in chronic gout also in members of gouty families, sometimes without acute form.

ALIMENTARY SYSTEM.—Dyspepsia, constipation, and pharyngitis common.

AFFECTIONS OF THE SKIN. *Eczema* common, especially of, and behind, ear. Pruritus and psoriasiform eruptions occur. Nails often brittle.

AFFECTIONS OF THE EYE. Itching of the eyeballs. Conjunctivitis. *Iritis*. Possibly glaucoma.

Retrocedent or Metastatic Gout.—During an acute attack, local condition may suddenly abort, while serious and even fatal symptoms appear:

i. CEREBRAL. Coma or delirium.
ii. GASTRIC. Pain, vomiting, and diarrhœa.
iii. CARDIAC. Præcordial pain, dyspnœa, tachycardia, and irregularities.

Complications and Sequelæ.—

RENAL. Chronic interstitial nephritis invariable in chronic cases. Uræmia may occur.

CIRCULATORY SYSTEM. Arteriosclerosis, myocarditis, hypertrophy of left ventricle common with usual symptoms. Thrombosis not uncommon, usually lower limbs. Pericarditis only with nephritis, high mortality.

PULMONARY SYSTEM. Emphysema and chronic bronchitis common. Asthmatic attacks occur.

GLYCOSURIA. Common in fat subjects, diabetic symptoms rare.

GRAVEL AND CALCULI. May occur, but most subjects escape. *Urethritis* not uncommon, may follow catheterization.

DIAGNOSIS.

In Acute Attacks. Usually simple. (1) *Arthritis*, often recurrent, in big toe or single joint. (2) Sudden onset. (3) Joint *swollen, shiny, red, and tender*. (4) Patient a full liver.

In Chronic Forms. More difficult. Consider (1) Patient's mode of living. (2) Character of early attacks. (3) Tophi. (4) Condition of joints.—X rays show deposits are peri-articular; (5) Condition of arteries. Prolonged analysis of urine may prove diminished excretion of uric acid.

Diagnosis from:—

ARTHRITIS DEFORMANS. Usually multiple joints from onset, osteophytic growths; wasting of muscles. In upper extremity, ulnar deflection, wasting of interossei muscles, Heberden's nodes. X rays show atrophy of bone.

RHEUMATIC FEVER.—Age under thirty. Fever higher. Attacks larger joints. Joints not red or shiny. Gout never causes endocarditis.

SYNOVITIS.—Gonorrhœal, pyæmic, and traumatic.

Gout, *continued.*

TREATMENT.

Clinical forms for purposes of treatment are: (1) Acute gout; (2) Chronic and irregular gout, involving the general treatment of a gouty individual.

Acute Gout.—

LOCAL TREATMENT OF LIMBS.—Elevate. Wrap in cotton-wool. Warm fomentations of sod bicarbonate (℥j to Oj), with tinct. opii ʒj. Cradle to support bed-clothes.

DIET.—Milk diet, custard, etc., until acute symptoms subside. No alcohol or meat extracts.

BOWELS.—Freely opened: pill at night with dose of salts in morning (*see* CHRONIC GOUT).

DRUGS.—*Colchicum* eases the pains and shortens the attack; mode of action unknown. Administration not to exceed four days, being powerful gastro-intestinal irritant. Well given with alkalis and aperients. Examples:

1. ℞ Vin. Colchici ♏xv + Mag. Sulphat. gr. xxx
   Pot. Citratis gr. xxx Aq. Menth. Pip. ad ℥j
2. ℞ Vin. Colchici ♏xv Aq. Menth. Pip. ad ℥j
   Mag. Carb. gr. x
   Two-hourly for 4 doses, then four hourly.

If necessary, replace by sodium salicylate subsequently.

GREAT PAIN AND SLEEPLESSNESS.—Barbitone or aspirin. Avoid morphia if possible.

Chronic or Irregular Gout. *General treatment of gouty individual.* Indications are to control the general and especially the purin metabolism. General lines of hygiene and diet are well established; experimental and scientific arguments for or against various drugs are to be accepted with caution.

GENERAL HYGIENE. Important: regularity and moderation. Daily exercise, not to be excessive for the stout. Regular meals: moderation in diet: usually more fluid and less alcohol. Daily bath, warm clothing; avoid chills. Regular motions.

DIET.—To be most carefully controlled. General reduction more important than discrimination concerning certain articles, but all 'rich' substances excluded. Ascertain patient's preferences and also his work and needs. Diet to be planned for the patient as well as for the disease.

PROTEIN.—Meat and fish allowable. Meat only once daily. A weekly meatless or purin-free day. Chicken, white meat, and fish best, also bacon: butcher's meat in moderation. *Exclude* articles rich in purins, especially sweetbreads, liver, rich meat soups and sauces, duck, goose, rich game, and salmon.

CARBOHYDRATES AND FATS. Definite restriction only necessary *if dyspepsia.* Butter and fats given freely; cheese moderately. Bread, rice, etc., porridge, and potatoes allowed, and sugar for sweetening tea, etc. *Exclude* rich pastry and sweets, boiled new potatoes.

VEGETABLES – Allowable: but *exclude* tomatoes, and usually cucumber and rhubarb.

FRUIT Give freely, especially early in day. *Exclude* strawberries and usually bananas.

TEA AND COFFEE These contain methyl-purins, but tea may be allowed *Exclude* strong coffee.

TABLE SALT Excluded by Roberts and replaced by KCl on grounds that blood, when deficient in NaCl, removes it from less vital fluids, including serous cavities, and will thus reduce sodium biurate deposition Theory unconfirmed.

FLUID A glass of water to be sipped first thing in the morning and at night Alkalis may be added as below

PURIN-FREE DIET Milk, white bread, potatoes, other carbohydrates, pure fats, eggs (First three contain minute negligible amounts of purin) Not necessary as routine

ALCOHOL *Entire abstinence preferable* If necessary, whisky or still white wine, e g, with cardiac weakness or evening exhaustion after long hours Draught cider allowable *Exclude* beer, champagne and other sparkling wines, and port, especially old in bottle

MEDICINAL TREATMENT Theoretical aims (i) To keep uric acid in solution, e g, by alkaline carbonates, (ii) To increase excretion of urates e g by salicylates, (iii) To dissolve uric acid e g, by piperazine.

ALKALINE SALTS General experience [illegible] erts these as valuable Can be given well as natural waters or potassium citrate gr xv for as a prescribing mixture in tumbler of water several times daily for prolonged periods Lithium carbonate or citrate gr v suggested owing to solubility of lithium biurate but therapeutic dose can have little effect

GUAIACUM In chronic gout with pains Not [illegible] given simultaneously Examples

℞ Tinct Guaiac Ammon [illegible] Sp Æth Nitris [illegible]
Pot Iodid [illegible] Aq [illegible] ad ℥j

Three times a day

℞ Guaiacol Carb, gr x in cachets, with —
℞ Pot Iodid gr x | Aq Menth Pip ad ℥j
Tinct Nux Vom [illegible]

Three times a day

Tincture of guaiacum must be freshly prepared unpleasant taste Administer for long periods

SALICYLATES Increase output of uric acid dosage etc, as in rheumatism

SPECIAL DRUGS designed to dissolve uric acid are numerous and reputation is usually ephemeral e g, piperazine but solvent action when in blood serum is very slight

Atophan (phenoquin), gr. xxxv daily for four day periods, increases output of uric acid novatophan may also be tried give alkalis also, separately Good results.

Urea, gr. xxx, t.d.s., in solution: is diuretic

Chronic Gout—Medicinal Treatment, *continued.*

A good prescription to be given for long periods is :—

℞ ℞ Mag. Sulphat. gr. xl | Sp. Ætheris Nitrosi ♏xl
Mag. Carb. gr. xxx | Aq. Menth. Pip. ad ℥j
Pot. Iodidi gr. iij |
t.d.s.

REGULATION OF THE BOWELS.—Essential. Best achieved by drugs at night acting on the liver, with a *saline aperient* in the morning.

DRUGS.—Calomel gr. j to iij. Euonymin gr. ss to ij. Podophyllin gr. ¼. Extract of hyoscyamus gr. j to iv. Pil. hydrarg. gr. iij to vj. Compound extract of colocynth gr. iss to iv. Iridin gr. j to iij. Pills may include two or three of above (especially hyoscyamus), e.g., pil. colocynth. et cal

℞ Calomel gr. j | Ext. Colocynth. Co. gr. iv

Or,

℞ Euonymin gr. ij | Pil. Colocynth. Co. gr. iss
Ext. Hyoscyami gr. j (Lind)

LOCAL TREATMENT OF JOINTS.—If much pain, as for acute gout. *For chronic joints*: *Massage*, lightly. *Radiant heat* baths and hot air. Electricity and cataphoresis. These treatments mainly necessitate spas or special facilities.

SPA TREATMENT.—Of benefit in chronic cases, owing to routine and regular life. Mineral waters drunk at their source possess advantage of radio-activity. Contra-indicated in acute gout, myocarditis, and great debility. Among others are :—

BRITISH SPAS.—Bath. Buxton. Harrogate. Llandrindod. Strathpeffer.

UNITED STATES.—Bedford. White Sulphur Springs. Saratoga.

FRANCE.—Aix-les-Bains. Contrexéville.

COMPLICATIONS :—

DYSPEPSIA.—Give alkalis and bitters. *Ferments* may be useful.

℞ Tinct. Nux Vom. ♏v | Spt. Chloroform ♏x
Sod. Bicarb. gr. x | Inf. Gent. Co. ad ℥j
Three times a day.

ECZEMA.—General treatment as for chronic gout. Local applications as in eczema. Sulphur waters good, e.g., Harrogate, Strathpeffer.

GLYCOSURIA.—Great restriction in carbohydrates and sugar only in severe cases and with diabetic symptoms.

CHAPTER LVIII.

CHEMICAL PATHOLOGY OF GOUT.

Two essential facts in gout **are established: (1) Presence of excess of uric acid in the blood; (2) Deposition of sodium biurate in articular and other tissues. No other point is beyond discussion, and neither**

of these is pathognomonic of gout, since they occur in other conditions. Wollaston, 1797, proved gouty deposits contained uric acid. Garrod, 1847, demonstrated presence of uric acid in gouty blood.

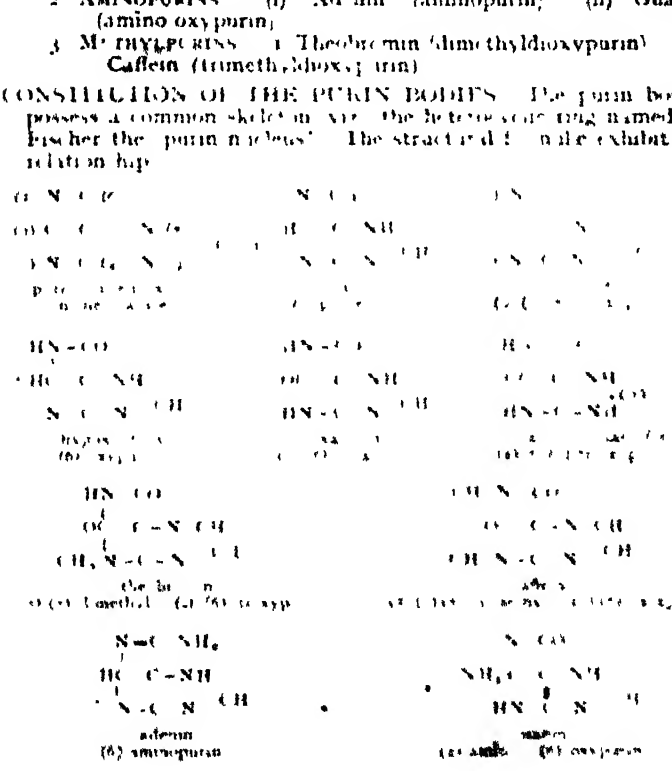
THE PURIN BODIES.

Abnormal metabolism of the 'purin bodies' produces the phenomena of gout. The occurrence of this abnormal metabolism is probably not immediately due to any abnormality of the purin bodies themselves, but to some remoter cause, possibly to some error in a protein with which they are normally combined when circulating in the blood, or to some error in the ferments concerned in their metabolism; of such questions practically nothing is known.

PURIN BODIES. Form three groups of which the following enter into human metabolism.

1. Oxypurins. (i) Hypoxanthin (monoxypurin). (ii) Xanthin (dioxypurin). (iii) Uric acid (trioxypurin, $C_5H_4N_4O_3$).
2. Aminopurins. (i) Adenin (aminopurin). (ii) Guanin (amino oxypurin).
3. Methylpurins. (i) Theobromin (dimethyldioxypurin). (ii) Caffein (trimethyldioxypurin).

CONSTITUTION OF THE PURIN BODIES. The purin bodies possess a common skeleton, viz. the heterocyclic ring named by Fischer the 'purin nucleus'. The structural formulæ exhibit the relationship



Gout—Chemical Pathology, *continued*

Source of the Purin Bodies Excreted in Man.—Two sources: (1) Exogenous; (2) Endogenous.

EXOGENOUS PURINS.—Ingested with food. Only certain foodstuffs affect purin excretion:—

(1) NUCLEIN-CONTAINING SUBSTANCES.—On decomposition these produce aminopurins, convertible into uric acid in the body. Specially abundant in the thymus (adenin) and pancreas (guanin).

(2) MUSCLE.—Contains the oxypurins xanthin and hypoxanthin, convertible into uric acid in the body. Specially abundant in meat extracts.

(3) CAFFEIN or THEOBROMIN (cocoa).—Methylpurins. *Increase purin excretion*, but *not convertible into uric acid in the body*.

ENDOGENOUS PURINS.—Arising from metabolism of tissues. Two main sources:—

(1) MUSCLE METABOLISM.—Produces xanthin and hypoxanthin. Increased by exercise.

(2) NUCLEIN.—Normally, and also in gout, of less importance than muscle. In leukæmia and leucocytosis, is origin of much uric acid.

DAILY EXCRETION.—*Purin nitrogen* in grammes: (1) Exogenous, 0·3 to 0·5; (2) Endogenous, 0·1 to 0·2.

Probably half the endogenous purins arising from metabolism are decomposed into urea, etc., in the tissues and do not reach the urine as purins. Total daily uric acid nitrogen about 0·5 grammes.

Formation of Purin Bodies and Uric Acid in the Tissues.—From nuclein both of food and of tissues by action of succession of specific ferments (enzymes): (i) *Nuclease*: frees adenin and guanin from nuclein. Distribution in tissues almost universal. (ii) *Deaminase*: converts adenin and guanin into hypoxanthin and xanthin. Distribution same as nuclease. (iii) *Oxidase*: converts hypoxanthin and xanthin (from any source—nuclein or muscle) into uric acid.

Most animals readily oxidize uric acid into (finally) CO_2 and NH_3 by the ferment *uricase*; the presence of this in man is unproved, and man, possibly owing to its absence, has difficulty in disposing of uric acid.

Variation in Purin Excretion in Gout.

IN CHRONIC GOUT.—(1) *Endogenous purins*: excretion about equal to lowest average of health, i.e., a constant diminution. (2) *Exogenous purins*: a purin rich meal to a gouty man causes an increased excretion, but smaller and slower than in health. (Thus retention occurs of purins from both sources.)

IN ACUTE GOUT.—Before and after attack, purin excretion is low. Shortly after attack commences, excretion rapidly *rises above normal limits*, then falls again.

Increase of Uric Acid in Blood in Gout.—In health, blood contains no *free* purins, and total is about 0·02 gm. per

1000 c.c. In gout: (1) Free purin (uric acid) is present, (2) Total amount is about 0·1 gram per 1000 c.c. Thus the uric acid in the blood is definitely increased. Theoretically, this increase may result from

1. *Increased production.* Against this: increased production, and increased amount in blood, occur in leukæmia and leucocytosis, and then are always accompanied by increased excretion.
2. *Diminished excretion by kidney, with normal production.* Supported by low purin excretion in gout and deficient excretion after purin rich meal. *Generally accepted cause.*

Diminished Excretion by Kidney of Uric Acid in Gout.—Two main possibilities to account for cause: (1) A primary renal defect, i.e., gout is a renal disorder. Against this: large excretion in acute gout proves that kidneys are not inherently unable to eliminate uric acid. Gout is not a primary renal but a metabolic disturbance. (2) Uric acid in gout circulates in an abnormal form which kidneys cannot eliminate. A reasonable working hypothesis.

The Form in which Purins Circulate in the Blood.—Two questions are involved:

1. Are the Purins combined with Proteins?—No test for uncombined purins reveals them in normal blood. Minkowski suggested that purins in blood are normally combined with protein from which the kidney can separate and excrete them. Von Noorden further suggests that in gout this protein is deficient (or abnormal), hence kidney has to deal with uric acid uncombined or abnormally combined and with difficulty excreted. This question deals with cause of retention of uric acid in gout.
2. In what Form is the Uric Acid?—Admittedly as a sodium salt. *Sir William Roberts' Theory.* Uric acid circulates as quadriurate $Na_4H_2U_4$, uric acid being a bivalent acid, written H_2U, comparatively soluble but unstable in blood; this changes slowly to biurate $NaHU$, stable but less soluble and therefore tending to be deposited in the tissues. Existence of quadriurate in blood is now disputed. *Gudzent's Theory* (modern modification of Roberts' theory). Uric acid circulates as biurate $NaHU$; biurate exists in two forms: (a) Unstable, soluble 'lactam' form, contains group $C(O)\cdots NH$— and (b) Stable less soluble 'lactim' form, contains group $-C(OH)\cdots N-$; on changing from (a) to (b), blood becomes a supersaturated solution, and biurate is deposited. (This question deals with cause of deposition of urates.)

   In leukæmia, etc., where also excess of urates occurs in blood, compensatory excretion does not allow time for the change in form, and hence no deposition follows.

Relation of Gouty Paroxysms to Deposition of Biurates.—Various questions arise:—

Does a paroxysm correspond with deposition of biurates?—

Gout—Chemical Pathology, *continued.*

Frequently illustrated by increase in size of visible deposits.

How does deposition excite a paroxysm?—Generally ascribed to inflammation resulting from the irritation caused by rapid deposition.

Selection of certain joints.—Ascribed to: (1) Frequent injury: in great toe, inflammation of joint is a common occurrence; forms focus for deposit. (2) High percentage of sodium salts, decreasing solubility of urates: is maximum around joints. (3) Low temperature, locally decreasing solubility.

Why are the urates suddenly deposited in gout?—Deposition of crystals from any supersaturated solution *in vitro* depends on various factors, many at present obscure, e.g., shaking, temperature, presence of a nucleus, increase of certain ions (in this case sodium). Such deposition *in vitro* may occur either *suddenly* or *gradually* in different conditions. A minute change may result in a sudden and complete deposition. Exact factors in gout unknown.

Rise of purin excretion during acute attack also unexplained.

Notes.—

EBSTEIN considered that initial change was necrosis of tissues near joints, due to an abnormal ferment. Not accepted.

ABNORMALITY OR DEFICIENCY OF FERMENTS (*see above*) Has been advanced as cause of excess or abnormal form of purins in gouty blood.

Action of these ferments little known, but undoubtedly important.

CHRONIC INTERSTITIAL NEPHRITIS AND GOUT. Often co-exist. In chronic nephritis, deposits of urates occur without paroxysms: excretion of urates is diminished and free urates often detectable in blood; deposits probably occur *slowly*. Chronic nephritis very common in chronic gout: possibly injury by circulating purins (Garrod).

Many essential questions in gout are yet unsolved, e.g., importance of ferments, influence of xanthin and other purins besides uric acid.

✓ Summary of Chemical Pathology of Gout.

(1) The uric acid and purins circulate in the blood in abnormal form.
(2) The kidney is unable to separate and eliminate uric acid from this combination.
(3) Uric acid salts consequently accumulate in the blood.
(4) These uric acid salts alter from a soluble to a less soluble state, and the blood becomes supersaturated.
(5) Sudden deposition of urates occurs from this supersaturated solution.
(6) Inflammation is excited mechanically in the tissues affected, and a gouty paroxysm occurs.
(7) After the deposition of urates and the resulting paroxysm, the blood is temporarily freed of the excess; the patient often feels unusually well. The unknown cause is still present, and the accumulation of urates in the blood commences again.

CHAPTER LIX.

DIABETES MELLITUS.

A condition due to chronic abnormality of the carbohydrate metabolism, and characterized pathologically by hyperglycæmia and by long continued glycosuria, and clinically by thirst, polyuria, emaciation, and tendency to coma.

Etiology.

AGE. All ages from birth commonest thirty to sixty years In youth, rapid and severe

SEX About 3 males to 2 females

RACE Hebrews and Eastern races very liable

HEREDITY. Of doubtful influence, but several cases may occur in one generation

PREDISPOSING CAUSES - Common in obesity. Excessive ingestion of carbohydrates of no obvious influence More common in upper classes and to some degree in neurotic persons

For metabolism of carbohydrates, normal and diabetic, for theories of diabetes, and for acidosis, *see* CARBOHYDRATE METABOLISM Chapter LX, p 323

Morbid Anatomy.- (Changes are mainly those of complications, except in pancreas.)

PANCREAS *See* p. 327.

LUNGS Tuberculosis or pneumonia common Rarely, gangrene

KIDNEYS Large markedly brick-red kidneys occasionally occur (diabetic kidney) Common change is hyaline degeneration in descending loop of Henle. Chronic nephritis frequent

LIVER Enlarged fatty degeneration common (Cirrhotic in hæmachromatosis) Glycogen absent

BLOOD -Rarely lipæmia, if death in coma

ARTERIOSCLEROSIS Common. Occasionally myocarditis

Clinical Classifications. On various bases —

1 (a) *Acute*, usually in youth (b) *Chronic* later life
2. (a) *Mild*, glycosuria only on carbohydrate diet (b) *Severe*, glycosuria on carbohydrate-free diet.

Symptoms.—

ONSET. —Gradual. Rarely sudden symptoms after shock

INITIAL COMPLAINTS.—Commonly (1) Thirst, (2) Polyuria; (3) Emaciation and weakness, (4) Boils, ulcers, and carbuncles. Occasionally: (5) Gangrene, (6) Pruritus; (7) Cataract.

CHARACTERISTIC FEATURES. —

THIRST } Fluid needed for excretion of sugar, and for
POLYURIA } hyperglycæmia.

APPETITE usually enormous. Digestion g.

Diabetes Mellitus—Symptoms, *continued.*

EMACIATION AND WEAKNESS—Usually rapid and progressive: extreme in youth.

TONGUE—Large, dry, and red ('raw beef').

SKIN.—Dry. Sweats occur with phthisis.

Temperature low. Pulse rapid. Constipation common.

Urine.—*Chief characters* (1) Amount: from 3 to 4 or more litres (100 to 150 ounces or more). (2) Specific gravity, usually 1025 to 1045. (3) Colour, very pale. (4) Dextrose present: mild cases, 1 to 2 per cent; severe, over 5 per cent. Daily excretion very variable: often 100 to 500 grammes (3 to 15 ounces), but may exceed this. (5) Albumin absent in mild forms; trace common in chronic and severe forms, and with chronic nephritis. *Other features*: Increased excretion of total N, uric acid, urea, phosphates.

Blood.—Red cells variable, secondary anæmia or polycythæmia. Rarely lipæmia. For 'Sugar in Blood,' *see* CARBOHYDRATE METABOLISM, p. 327.

Complications.—

1. DIABETIC COMA AND ACIDOSIS. Cause of death in 40 to 50 per cent, in youth yet higher. Progress often rapid, may be no warning. Odour of acetone in breath.

   PRODROMAL SIGNS. (1) Epigastric pain, (2) Anorexia, (3) Restlessness or headache. Also acetonuria, rise of NH in urine, numerous casts, fall of alveolar CO_2, fall of plasma bicarbonate.

   CLINICAL TYPES. (1) *Dyspnœic coma.* Common form. Slow deep respirations (Kussmaul's 'air hunger'), feeble pulse, rapid coma. (2) *Alcoholic type.* Headache severe, thick speech, no dyspnœa, gradual deepening coma. (3) *Type with collapse.* Lividity, shallow rapid respiration, deepening coma. Differentiation of type often indefinite.

2. PULMONARY DISEASE. Cause of death in 40 per cent. (i) Tuberculous caseating bronchopneumonia, rapid. (ii) Acute pneumonia. Rarely (iii) Gangrene.

3. SKIN AND SUBCUTANEOUS LESIONS. Very common, attributed to sugar in blood. (i) Boils and ulcers. (ii) Carbuncles. (iii) Itching, general, or near urethra. (iv) Diabetic gangrene: rare under 50 years, begins at toes, usually moist, blue discoloration, extends. Arteriosclerosis present in area. Glycosuria often slight.

   Rare: Pigmentation (in hæmachromatosis).

4. PERIPHERAL NEURITIS.—Tinglings common. Absence of knee-jerks first sign. Perforating ulcers not uncommon. 'Diabetic tabes': knee-jerks absent, steppage gait, pains in legs.

5. EYE.—(i) Cataract: in young or old, rapid, often bilateral, soft type. (ii) Diabetic retinitis. Changes are (a) glistening patches, often near macula, but not in star shape as in albuminuria, (b) small hæmorrhages. The two changes occur separately.

or together. Distinction from albuminuric retinitis rarely definite. (iii) Rare: Amblyopia, blindness, central scotoma, from retrobulbar neuritis. Thrombosis of central vein. Optic atrophy. Sudden amaurosis. Lipæmic vessels.

6. RENAL.—Chronic nephritis not uncommon in chronic cases. Albuminuria otherwise uncommon, except in severe and chronic cases. Occasionally: cystitis, pneumaturia (yeast in bladder). When acute nephritis occurs, sugar in urine temporarily diminishes or disappears, from impermeability of diseased kidney.

7. PREGNANCY.—Pregnancy rare; abortion usual; disease becomes aggravated.

OTHER COMPLICATIONS.—Gastritis, diarrhœa, myocarditis, arteriosclerosis.

Prognosis.—Main factors are:

AGE.—In young, rapidly fatal, usually in few months, limit 1 to 2 years. In older patients may be chronic, especially with obesity; duration many years.

GRADES OF SEVERITY.—(1) Mild: on carbohydrate-free diet, no glycosuria. (2) Severe: on similar diet glycosuria persists. (3) Ketosis: if unchecked by treatment prognosis very grave.

Terminations (see "Complications"). In youth, almost invariably coma. In older group, coma and lung complications about equal. Occasionally, gangrene, sepsis, nephritis, etc.

Diagnosis of glycosuria depends on examination of urine. Further distinctions necessary: (1) True diabetes: sugar present in urine over long period, with hyperglycæmia (rarely absent). (2) Transient and non-diabetic glycosuria: occurs in obesity, certain conditions of pituitary and thyroid gland, rarely in acute fevers, and carbohydrate excess.

Dextrin occasionally by cane sugar or lactose (no fermentation with yeast); rarely by glucose.

Theoretical, but very rare, difficulties are: alkaptonuria, pentosuria.

Treatment.—Basis of all treatment is dietetic reduction in of carbohydrates, secondly of fats and proteins, in order to lower the hyperglycæmia and the acidosis. Method much influenced by Allen's fasting treatment.

NOTE.—All changes in diet must be effected gradually. Any sudden change may evoke acidosis and coma. Amount of sugar passed in urine should be estimated daily, and, if possible, blood sugar twice a week.

CLASSIFICATION OF SEVERITY. Place on a "test diet" (Hutchison) of meat, eggs, green vegetables, butter, and 4 oz. of bread. Groups: (a) *Mild*: excretion of glucose less than 70 grm. (the yield of 4 oz. of "toast"). (b) *Severe*: excretion exceeds 70 grm. In this group ketonuria is often present.

GENERAL TREATMENT.—Warmth. Regulate bowels. Avoid chills. Moderate exercise. Treat all sources of infection, e.g., boils and oral sepsis. Avoid general anæsthetics.

Diabetes Mellitus—Treatment, *continued*

FASTING TREATMENT (Allen, Joslin).—Two objects: (1) To abolish glycosuria and acidosis; (2) To find lowest level of diet on which patient can maintain nutrition, just sufficient to live in comparative comfort. Method aims at keeping patient thin and underfed, the pancreatic secretion often being just sufficient to maintain such metabolism. The diet fixes and limits fats and proteins as well as carbohydrates.

PRELIMINARY. Patient placed in bed, and diet gradually reduced. Then three stages:

1. FASTING. No food until glycosuria and acidosis abolished. Duration 2 to 5 days. Fluid unstinted. If more than 2 days necessary, give 300 c.c. meat broth daily. Also give *alcohol*, whisky or brandy.
2. FIXING DIET.—Careful records kept of patient's weight, urine, and diet. All diet to be measured. Commence when glycosuria and ketonuria have been absent for 24 hours. Gradual addition of (a) Carbohydrates: small amounts at first by vegetables of low carbohydrate grade, then through increasing grades and amounts, finally to potatoes and bread if urine remains sugar-free. The limit of carbohydrate tolerance is the amount on which sugar commences to appear in the urine. The carbohydrate diet next should contain not more than two thirds of this quantity. (b) Proteins: when urine is sugar free for 2 days add 3 eggs, then gradually give meat, 5 grm. added daily up to 1 or 1.5 grm. protein per kilo. body weight. (c) Fats: when above diet has been progressing add 25 grm. fat daily (butter, bacon) until weight is no longer lost.
   - If sugar returns give a fast day, and recommence with lower diet. Give fluid freely throughout.
   - The final diet should contain 35 calories per kilo. body-weight. If this is well tolerated and more is demanded increase amount carefully, altering one constituent at a time, commencing with fat.
3. SUBSEQUENT TREATMENT. Treatment, as standardized to be maintained. One day weekly a fast day, or vegetable day with greatly reduced carbohydrates in milder cases.
   - With *ketonuria* always give sodium bicarbonate in addition to fasting.

COMA.—When coma threatens:

1. FASTING TREATMENT to be commenced at once.
2. ADMINISTRATION OF SODIUM BICARBONATE. (a) By mouth, ʒj hourly. (b) By enemata, ℥j in ℥x water, four hourly. (c) Intravenously in severe cases, sterile solution of normal saline with 2 per cent sodium bicarbonate added, 2 pints injected four to six hourly.

CARBOHYDRATE-FREE DIET.—When 'fasting treatment' is not being adopted (a) Place, by gradual reduction, on a carbohydrate-free diet. Articles: meat, eggs, fish, green vegetables,

bacon and diabetic bread (carefully selected). Urine may become sugar-free in few days. (2) Maintain for two weeks on above diet after sugar disappears. (3) White bread, one ounce, added to diet, increasing one ounce every third day till sugar appears; of final amount two-thirds may then be allowed daily.

In subsequent treatment, fast or vegetable day once weekly.

If above treatment fails to render urine sugar-free, adopt fasting treatment, and invariably if ketonuria is present.

CHAPTER IX.

CARBOHYDRATE METABOLISM, NORMAL AND PATHOLOGICAL.

In carbohydrate metabolism there are two main considerations:

1. Control of carbohydrate metabolism by the body and the influence of the ductless glands.
2. Chemical phenomena of carbohydrate metabolism, normal and abnormal.

The liver and muscles represent laboratory and engineering plant for the preparation and use of carbohydrates. The ductless glands are the workers in charge.

The urinary sugar in diabetes is dextrorotatory dextrose, also known as glucose.

Diabetes is a chronic disorder of carbohydrate metabolism.

Influence of Ductless Glands and other Tissues.—

The ductless glands form two groups: (a) *Inhibitors* of glycogenolysis (output of dextrose into the blood): pancreas, parathyroid (minor influence). (b) *Intensifiers* of glycogenolysis: suprarenal, pituitary and thyroid (minor influence). These groups have opposite actions, but classification based on glycogenolysis is of doubtful accuracy.

PANCREAS. Hyperglycæmia occurs in absence of internal secretion of pancreas (the external secretion has no influence). Together with suprarenal, form essential control of carbohydrate metabolism.

Mode of Action. Principal theories are: (a) Pancreatic hormone, retards glycogenolysis by liver, i.e., 'inhibitory' to liver. (b) Pancreatic hormone is necessary to enable muscles to combust and utilize dextrose. Theory that muscles need pancreatic hormone to combust carbohydrates originally suggested by Cohnheim from experiments *in vitro*; lately revived by perfusion experiments by Knowlton, Starling, and Lovatt Evans; *now widely accepted*. On this very probable theory, it becomes incorrect to term pancreatic secretion an 'inhibitor' of glycogenolysis, its action being primarily on the muscles and not on the liver.

Carbohydrate Metabolism, *continued*

SUPRARENAL GLANDS.—Action of adrenalin :—

1. Hyperglycæmia and glycosuria follow injection, even in starving animals.
2. Action specially marked in diabetes and depancreatized animals
3. In Addison's disease, where internal secretion is deficient, hypoglycæmia and increased carbohydrate tolerance are present (also in suprarenal extirpation)

Mode of action Unknown Possibly increases glycogenolysis by liver or acts on the muscles.

Opposite to action of pancreas.

PITUITARY GLAND (HYPOPHYSIS) Note (a) With hypersecretion of posterior lobe, hyperglycæmia and glycosuria occur, as in early acromegaly and certain tumours of gland (b) With hyposecretion of pituitary, hypoglycæmia and increased carbohydrate tolerance occur, as in late acromegaly Frohlich's disease, and removal of gland In fractures of skull and operations disturbing gland, hyperglycæmia is not uncommon

Mode of action —Supposed to inhibit pancreas

THYROID GLAND —Note (a) With excessive dosage hyperglycæmia and glycosuria result Not uncommon in Graves disease (b) In myxœdema, hypoglycæmia and high carbohydrate tolerance are present

Mode of action Supposed to inhibit pancreas

PARATHYROID GLANDS Opposite action knowledge incomplete

NERVOUS SYSTEM Claud Bernard's piqure', puncture of floor of 4th ventricle results in glycosuria Action is through left splanchnic nerve to the suprarenals, *increasing adrenalin in blood* Glycosuria ceases when liver is emptied of glycogen Cerebral tumours, fractures, etc may produce glycosuria also from action on pituitary gland

KIDNEYS Hyperglycæmia accompanies (in predisposed) practically all glycosuria in man hence *glycosuria through kidneys is not a cause* of diabetes mellitus Diseased kidneys excrete sugar less readily than in health, e.g. in acute or chronic nephritis with acute nephritis occurring in diabetics glycosuria may temporarily cease in diabetics, kidneys also less *permeable to sugar than in health* (Graham's high leak point)

PHLORIDZIN DIABETES Exceptional produced experimentally by administration of phloridzin as glucoside Hyperglycæmia occurs. Excretion of sugar enormous continues on carbohydrate free diet and when liver empty of glycogen hence sugar is produced from protein Mode of action unknown possibly kidney can split off and excrete glucose from phloridzin, which then re-combines with blood-sugar, forming a cycle

Chemistry of Carbohydrate Metabolism.—

CARBOHYDRATES Those concerned are

1. MONOSACCHARIDES $C_6H_{12}O_6$ (i) Dextrose (glucose, grape-sugar) ; (ii) Lævulose (fruit sugar), (iii) Galactose, (iv) Mannose.

2. DISACCHARIDES — $C_{12}H_{22}O_{11}$: (i) Saccharose (sucrose, cane-sugar); (ii) Lactose (milk sugar), (iii) Maltose.
3. POLYSACCHARIDES — $(C_6H_{10}O_5)_n$: Starch, cellulose, glycogen, dextrin.

Reactions: *Fermentation with yeast*: monosaccharides. *Fehling's solution* reduced by monosaccharides, lactose and maltose.

Normal Carbohydrate Metabolism.

IN ALIMENTARY CANAL. Action on ingested carbohydrates: (i) *Starch* converted finally into maltose and dextrin by diastatic ferments of saliva, pancreatic and intestinal juice; (ii) Maltose and dextrin (ingested or from previous stage) converted into *dextrose* by ferments in intestinal mucous membrane; (iii) Cane-sugar into dextrose and lævulose; (iv) Lactose into dextrose and (possibly) galactose. All these carbohydrates thus *become monosaccharides, are absorbed, and pass to the liver*.

IN LIVER. Processes are:
1. Monosaccharides arriving in the blood are converted into glycogen.
2. Glycogen thus formed is converted into dextrose and passed into blood as required by tissues. *Dextrose is sole carbohydrate used by tissue liver*.
3. Glycogen in excess stored in liver. Maximum about 150 grm.

IN THE TISSUES. Processes are:
1. Combustion of dextrose and production from proteins essential fatty acids of carbohydrates.
2. Storage of excess as glycogen: maximum for body about 150 grm.

Storage of excess carbohydrates: is glycogen thus stored in (1) liver (2) muscles. Total about 300 grm.

SUGAR IN THE BLOOD. See METABOLISM IN DIABETES, p. 327.

SOURCES WHENCE LIVER CAN PRODUCE DEXTROSE:
- CARBOHYDRATES: Ingested.
- PROTEINS: From diet and body tissues. Proof: (1) from result of diet in severe diabetes, viz., glycosuria (a) continues on carbohydrate free diet; (b) increases with amount of protein in diet.
- FAT: In diabetes, glycosuria is uninfluenced by amount of fat in diet, hence generally believed that fat is not converted into sugar. See FAT METABOLISM, p. 329.

Carbohydrate Tolerance. 'Assimilation Limit'.

Method of Estimation: Dissolve 100 grammes glucose in 250 c.c. water, and give in the morning on empty stomach. Glycosuria proves diminished tolerance.

Normal Assimilation Limits (single dose on an empty stomach): — Glucose, cane-sugar: 150 to 200 grm. Lactose: 120 grm. or less. These are lower limits: most people can assimilate more.

Carbohydrate Metabolism, *continued.*

Variations in Carbohydrate Metabolism.—

Abnormality may arise from variation of supply and demand, or from perversion of metabolism.

SUPPLY DEFICIENT, or less than demand of muscles.—Sugar in blood remains constant (reduced in excessive exertion), maintained from glycogen stores, and later from protein.

SUPPLY EXCESSIVE. (1) *Temporary and moderate*: excess stored as glycogen. (2) *Persistent and moderate*, excess converted into, and deposited as, fat, i.e., obesity results. Blood-sugar constant in these forms. (3) *Great excess*: hyperglycæmia results, and glycosuria, i.e., *alimentary glycosuria.*

PERVERSION OF METABOLISM.—Gives rise to *diabetes*, and glycosuria associated with diseases of ductless glands.

TRANSIENT GLYCOSURIA.—Alimentary glycosuria. Occurs in:—

1. NORMAL PERSONS. Sudden great excess of sugar. *Starch never causes glycosuria in normal persons*, however great its ingestion, owing to slowness of absorption and digestion.
2. LOWERED 'CARBOHYDRATE TOLERANCE'.—In *obesity*, alimentary glycosuria after ingestion of sugar occurs easily: usually ascribed to glycogen and fat depots being overfull. In absence of obesity, occurrence reveals severe pancreatic disease or conditions of ductless glands (associated with glycosuria: thyroid, pituitary, suprarenal, and cerebral lesions), or a low 'renal threshold'. Rarely in acute fever.

'Alimentary glycosuria' is distinguished theoretically from diabetes by never occurring after ingestion of starch, but group (2) merges into, and some cases may develop, true diabetes.

Summary of Normal Carbohydrate Metabolism.

1. *Carbohydrates ingested* are converted into monosaccharides by ferments in the intestinal juices or mucous membrane and are then absorbed.
2. *In the liver*, the monosaccharides are converted into glycogen, sufficient to maintain blood sugar at required level is reconverted into dextrose, and the excess stored as glycogen.
3. *Blood* acts purely as means of transport and communication, and the kidneys as safety-valves when blood sugar rises too high.
4. *The muscles* utilize carbohydrates for production of energy, storing temporary excess as glycogen.
5. As blood-sugar falls (from muscular exertion), liver supplies deficiency, first from glycogen and then from protein of food and tissues.
6. If ingestion of carbohydrates be constantly excessive, unused balance is converted into, and deposited as, fat.
7. *Ductless glands* control the above processes in general, especially the pancreas (inhibiting sugar output from liver) and the suprarenals (accelerating sugar output).

METABOLISM IN DIABETES.

Important phenomena are connected with: (1) *Disease of the pancreas*; (2) *Sugar in the blood*; (3) *Metabolism in the liver*; (4) *Metabolism in the muscles*; (5) *Glycosuria*.

1. Relation of Pancreatic Disease to Diabetes (*see also* INFLUENCE OF DUCTLESS GLANDS, p. 323).—Minkowski and von Mering discovered the relationship.

MORBID ANATOMY OF PANCREAS IN DIABETES.—

i. Chronic interacinar pancreatitis present in at least 70 per cent, with atrophy of the islands of Langerhans (Opie).

ii. Carcinoma, lipomatosis, interstitial pancreatitis in a few cases, but cause glycosuria only when very advanced.

iii. Normal in 10 to 15 per cent. Possibly non-pancreatic diabetes.

EXPERIMENTAL.—Diabetes follows removal of not less than four-fifths of pancreas. External secretion has no influence.

CONCLUSION.—Islands of Langerhans produce internal secretion essential for prevention of diabetes.

2. Sugar in the Blood. Hyperglycæmia, or abnormal amount of blood-sugar, is essential factor in occurrence of diabetic glycosuria, though various factors affect the amount excreted. Blood-sugar estimations and study of curve are especially of value in assessing importance of slight glycosuria.

A. BLOOD SUGAR IN NORMAL MAN.—The amount varies considerably in different persons, but the limits may be taken at 0·05 to 0·12 per cent, with usual average of 0·08 to 0·10 per cent for the whole quantity in blood. McLean's method and Bang's titration method give results about 25 per cent higher, with an upper limit of 0·15 per cent. In a given person the amount remains constant, even with muscular exertion, unless strain or nervous shock or convey need for sugar to the liver by unknown method. In healthy persons glycosuria occurs automatically when blood sugar reaches 0·16 or 0·17 per cent; this point is known as the 'renal threshold'.

B. NORMAL BLOOD SUGAR CURVE AFTER A MEAL CONTAINING CARBOHYDRATES.—Blood sugar commences to rise within 5 minutes, reaches maximum in 30 minutes, then falls rapidly to normal or 'post' below, in 1½ hours, or 2 hours if carbohydrates were large in amount. Maximum is 0·16 to 0·17 per cent, viz. near normal 'renal threshold', at this point storage processes possibly come into action and reduce blood sugar to standard level (McLean and de Wesselow).

C. BLOOD SUGAR CURVE IN DIABETES.—

a. When not under diet, the level is above normal, i.e., hyperglycæmia. In moderate cases is below a normal 'renal threshold', but in severe cases is above, and may be 0·30 or higher.

b. After carbohydrate ingestion, variations from normal curve

Metabolism in Diabetes, *continued.*

are: (1) Rise persists longer and reaches higher level. (2) *Fall is slower and does not return to former level under 5 hours.*

c. After dieting (starvation), the level may be at or near normal. After a carbohydrate meal, the rise now may not exceed the 'renal threshold' and hence no glycosuria occurs, but the *slow fall remains.*

The following points may be noted:—

(1) Occurrence of glycosuria depends on the blood-sugar rising above the 'renal threshold', temporarily or permanently.

(2) The 'renal threshold' may be higher than normal. In chronic diabetes it tends to rise, and may be 0·20 or higher, the kidneys becoming injured and less permeable to sugar. Thus definite hyperglycæmia may be present without glycosuria.

Diseased kidneys excrete less readily than in health; with acute nephritis occurring in diabetes, glycosuria may temporarily cease.

(3) The amount of sugar in the urine does not run parallel to the degree of hyperglycæmia or to its variations. Hence the amount of sugar in the urine depends to some extent on other factors than the amount of sugar in the blood. Thus, as an important example, the percentage of sugar excreted, as well as the total amount, usually rises with the volume of urine; the excretion of sugar is hence affected by administration of diuretics. The existence of such factors obviously complicates the investigation of diabetes and the interpretation of results.

(4) '*Renal diabetes*'. The 'renal threshold' may be lower than normal and hence transient glycosuria occurs after a meal. Distinction from true diabetes rests on occurrence of a normal blood-sugar curve after carbohydrates. The glycosuria is slight (usually a few grammes a day), not accompanied by diabetic symptoms, and is affected or controlled by diet. Cases have been watched for many years without advance.

Diagnosis of 'renal diabetes' is only justified by careful blood-sugar tests; prognosis at present should be guarded, as some apparent cases have subsequently developed diabetic symptoms.

3. Metabolism in the Liver.—Glycogen is absent from the liver. Overproduction of sugar by the liver in diabetes is proved by the excretion of 10 to 30 pounds daily in some cases. The liver is not primarily at fault since: (a) Pancreatic disease is the predominant lesion; (b) Advanced liver disease is not associated with glycosuria (though levulosuria may occur).

4. Metabolism in the Muscles.—Glycogen is absent from the muscles. The muscles normally combust carbohydrates and produce energy from them; in diabetes the muscles are unable to do so, this inability being ascribed to absence of pancreatic hormone (*see* p. 323, INFLUENCE OF DUCTLESS GLANDS). Evidence exists that the muscles attempt to use fat and possibly protein.

CONCLUSION.—In the muscles in diabetes there is (1) Diminished consumption of sugar; (2) Abnormal metabolism of fat, especially in severe cases; (3) Absence of glycogen.

5. **Glycosuria.**—The characteristic outward sign of diabetes. Occurs automatically when blood-sugar exceeds 0·17 per cent (*see* SUGAR IN THE BLOOD, p. 327).

Protein Metabolism.—The protein metabolism is excessive, being used by the liver to produce sugar: this katabolism results in weakness and wasting. Excretion of nitrogen often rises to 20 or 30 grammes daily.

Fat Metabolism.—The metabolism of fat is abnormal, but at present very obscure. In acidosis it is an essential factor: lipæmia may even occur.

IS FAT CONVERTED INTO SUGAR IN DIABETES? Administration of fat has little, if any, influence on amount of sugar excreted, hence it has been long believed that fat is not converted into sugar. But such conversion might occur without affecting sugar in urine, or in blood, and is upheld by modern authorities on following grounds:—

1. *Respiratory Quotient.* When dextrose is completely combusted, CO_2 output equals oxygen intake, i.e., respiratory quotient $\frac{CO_2}{O_2} = 1$. For higher fats quotient is about 0·7, and lower for proteins. Normally it is 0·9: in diabetes it falls to a figure varying with severity down to 0·7. This indicates that energy is obtained not solely from sugar but also from fats, and possibly protein.
2. *The 'D to N Ratio'* (viz., ratio of dextrose to nitrogen excreted in grammes). From 100 grm. of protein (= 16 grm. N) 130 grm. dextrose is maximum procurable, based on carbon present, giving D to N of 8. In severe diabetes on strict diet, ratio is constant at 2·8 or rarely at 3·0; but a few cases are recorded with a ratio of 8, and are not unreasonably claimed as proof of production of dextrose from fat (Graham and Poulton).

General Theory of Diabetes.

1. Muscles are unable to use dextrose in blood, and send call to liver for more sugar.
2. Absence of action of pancreatic hormone causes this inability.
3. Liver is able to form, but does not use and retain, glycogen, which is at once reconverted into dextrose, and passed into the blood.
4. Liver responds finally by producing dextrose from protein of diet and the tissues.

Pancreatic disease is thus regarded as primary origin of diabetes. In absence of its hormone, muscles lack the power to combust carbohydrate. Unable to distinguish between carbohydrate which they cannot utilize and dextrose which does not exist, muscles send forth increasing calls which result in increased sugar production by liver, and consequently in hyperglycæmia, glycosuria, and the condition of 'diabetes mellitus'.

Metabolism in Diabetes, *continued.*

Non-Diabetic Glycosuria.

Not all glycosuria is diabetes mellitus. Various types described:

1. GLYCOSURIA due to disease of ductless glands other than pancreas.
2. TRANSIENT GLYCOSURIA. *See* p. 326.
3. RENAL GLYCOSURIA.—*See* p. 328.
4. DIABETES INNOCENS.—A non progressive glycosuria. May be familial. At present not accurately differentiated from renal type.

   The innocence or non diabetic nature of a chronic glycosuria, though it need not necessarily be rejected, should yet be decided upon with great caution.

Acidosis in Diabetes.

(Scarcely any point in 'acidosis' is yet beyond controversy.)

Acidosis signifies the presence in the blood of certain acid (acetone or ketone bodies). In the urine NH_3 is increased, and acetone bodies may be present; alveolar CO_2 is diminished. Acidosis is in general applied to the presence in the urine of acetone, diacetic acid, and β oxybutyric acid ('acetone bodies'); this condition has until lately been termed 'ketonuria' by Poulton.

Origin of Ketone Bodies.—(1) From *fat*: main source in diabetes; (2) From *protein*: probable but not proved.

Theories of the abnormal production of these bodies:

1. VON NOORDEN: *Acetone bodies are produced when metabolism of fat occurs in absence of sufficient metabolism of carbohydrates.* Ascribed to 'deficient oxidation' of fats. The successive steps in fat metabolism by normal oxidation are believed to be: (a) butyric acid, (b) β oxybutyric acid, (c) acetic acid, CO_2 and H_2O. With deficient oxidation the steps are expressed thus: (a) butyric acid $CH_3 \cdot CH_2 \cdot CH_2 \cdot COOH$, (b) β oxybutyric acid, $CH_3 \cdot CHOH \cdot CH_2 \cdot COOH$, (c) acetoacetic acid, $CH_3 \cdot CO \cdot CH_2 \cdot COOH$, (d) acetone, $CH_3 \cdot CO \cdot CH_3$.
2. POULTON: In diabetes *fat is converted into sugar and acetone bodies are produced during this abnormal metabolism.*

   Production of ketone bodies probably also occurs in protein through amino acids, e.g., leucin, constitution of which is closely allied to fatty acids.

General Theory of Acidosis.—Acidosis is the result of abnormal metabolism of fatty acids, or of allied bodies occurring in the disintegration of protein, such metabolism resulting in the production of ketone bodies.

In diabetes, this abnormal metabolism occurs when the muscles unable to make use of sugar, attempt to utilize fat or even sugar, or it may possibly take place in the liver.

Compensatory Action of Body. The tissues attempt, in slighter stages successfully, to neutralize the acids first by the alkalis (e.g., K, Na) already present, secondly by production of ammonia.

instead of conversion into urea, large amounts of NH_3 combine with the acids, hence great rise in excretion of NH_3.

Mode of Production of Symptoms.—May be (1) Direct toxic action of β oxybutyric acid; (2) Acid intoxication (Stadelmann). Neither acetone nor diacetic acid is toxic for *healthy* animals. (Davy ascribed coma to CO_2 narcosis, resulting from acidity of blood causing lower carrying power, and hence accumulation of CO_2 in the tissues.)

Excretion. Ketone and diacetic acid precede β oxybutyric acid in urine, and disappear first. Exception of β-oxybutyric acid may reach 100 to 200 grm. daily, of acetone 50 grm.

Tests for Acidosis.—

I. URINE.—

(A) *Presence of ketone bodies.*—(a) Acetone. Rothera's sodium-nitroprusside test. (b) Diacetic acid. Gerhardt's ferric chloride reaction. (c) β oxybutyric acid. no simple test.

Note.—Ferric chloride a fairly sensitive test for diacetic acid (positive with 1 part in 5000), but does not react with acetone. Rothera's test reacts with acetone, but [illegible] test for diacetic acid (1 part in 400,000). positive test is usually due to latter, and is no proof of presence of acetone, even if ferric chloride reaction is negative.

Acetone in urine is largely produced by decomposition of diacetic acid subsequent to excretion by kidneys.

(B) *Lack of ammonia.*—Of total urinary N, about 3 per cent. is normally excreted as NH_3. Above 5 per cent. suggests acidosis, when symptoms are present, is often 20 to 30 per cent. or higher. *Acid precedes urinary casts.*

(C) Casts in urine suddenly numerous ('coma casts').

II. LOW PERCENTAGE OF ALVEOLAR CO_2 BONDIOXIDE.—Normally from 4.5 to 6.2 per cent. In coma falls to 2 per cent. By Ruderman's method estimation at bedside is rapid and simple.

III. BICARBONATE IN BLOOD PLASMA. Falls in acidosis. The fall in alveolar CO_2, and also in plasma bicarbonate precedes the appearance of ketonuria, and hence is the earliest warning of acidosis.

CHAPTER XVI

DIABETES INSIPIDUS.

A rare chronic affection characterized by the passage of large amounts of urine of low specific gravity, and free from sugar and albumin. Two groups:—

Primary or Idiopathic Group.—No organic lesion. Hereditary factor marked: may be through many generations. Usually males.

Diabetes Insipidus—Primary, *continued.*

ONSET frequently in youth Slow and insidious.

ORIGIN probably renal, an inability to secrete concentrated urine thus NaCl administered increases volume, but not specific gravity, of urine.

Secondary or Symptomatic Group.—Usually, possibly invariably, a lesion diminishing formation of, or obstructing passage into circulation of, secretion of posterior lobe of pituitary this secretion diminishes excretion of urine May result from (1) Syphilis, common, basal meningitis involving pituitary (2) Tumours and other lesions of pituitary. (3) Organic disease of central nervous system affecting circulation of cerebrospinal fluid, e g, blockage of foramen of Magendie as a sequel of cerebrospinal fever. (4) Trauma. (*See also* DISEASES OF PITUITARY BODY)

ONSET usually gradual. in trauma, onset often sudden and immediate

Action may be vasomotor disturbance of kidney, since polyuria also occurs occasionally in abdominal tumours, tuberculous peritonitis, injuries to spine.

Morbid Anatomy.—In primary group, no characteristics bladder and ureters may be hypertrophied

Symptoms.—

(1) *General health* good, *patient usually thin but not emaciated* (2) *Polyuria*, (3) *Thirst* (4) *Appetite* usually normal Constipation Skin dry Temperature subnormal Blood normal

URINE —Amount, often 10 to 20 litres (350 to 700 ounces) Specific gravity, 1001 to 1005 Almost colourless Sugar and albumin absent, or trace of albumin in late stage Urea and normal constituents in low percentage daily excretion of urea, etc, variable Inosite (muscle sugar) occasionally present, possibly 'washed' out from muscles by rapid passage of fluid

In secondary group, various other signs due to cerebral tumour etc Occasionally trace of sugar from affection of pituitary gland In syphilitic cases, transient temporal hemianopia common

Prognosis.—In primary group often long life, death occasionally from pneumonia, or from alcoholism, though alcoholic tolerance is high (probably from dilution)

In secondary group, depends on lesion.

Diagnosis.—From

1. DIABETES MELLITUS —By *absence of glycosuria and of hyperglycæmia*
2. CHRONIC NEPHRITIS —By absence of albumin casts, and arteriosclerosis.
3. HYSTERICAL AND FUNCTIONAL POLYURIA—This is transient or intermittent other signs of hysteria present
4. PITUITARY DISEASE, cerebral tumour, etc, require careful examination.
5. WASSERMANN REACTION to exclude syphilis.

Treatment.—If syphilitic, usual treatment. In other cases, treatment unsatisfactory. Polyuria can be reduced by *injections* of pituitary extract, but action transient.

FLUIDS.—Gradually reduce, but not after urine ceases to fall in amount.

DIET.—A salt free diet with low protein content should be tried. Includes eggs, fish, rabbit, butter, fruit, vegetables, rice.

DRUGS.—Doubtful value. Ergot, liquid extract ♏ x to xx, t.d.s. zinc valerianate gr. xv to xxx, t.d.s.

CHAPTER LXII

OBESITY AND OTHER LIPOMATOSES.

OBESITY.

An excessive deposit of fat. The condition is only a symptom, and the causes are various.

Etiology.—

RACE.—Common in certain races, especially Eastern.

HEREDITY.—A definite factor. Often associated with gout.

SEX.—Females predominate, possibly partly connected with internal secretions.

Pathogenesis.—Essentially lack of proportion between intake and combustion in the organism. Causes include: (1) *Excessive intake*, usually but not invariably combined with inactivity. Heavy eaters, especially of carbohydrates and fats. Heavy drinkers, especially of beer. (2) *Internal secretions*. Little understood. (3) *Sexual*. Increase of fat common at puberty, after castration, after the menopause, during pregnancy and lactation. (4) *Pituitary gland*. Secretion controls carbohydrate and fat metabolism. Tumours are associated with Frohlich's 'dystrophia adiposo-genitalis', characterised by adiposity and sexual infantilism (*see* PITUITARY GLAND). Obesity in young may be due to deficient pituitary secretion.

Morbid Anatomy.—Great increase of fat in all sites where normally found, e.g., subcutaneous, omentum. Fatty infiltration of heart invariable. Various complications.

Symptoms and Complaints.—(1) *Increased size*, alteration of personal appearance. (2) *Sweating*. (3) *Various symptoms of fatty myocarditis*, viz., shortness of breath, cardiac pains and weakness. (4) *Sleepiness*. General health may be good.

Two general types sometimes distinguishable: (a) Plethoric; (b) Anæmic—especially women.

Obesity, *continued*

Complications.—(1) From muscular weakness, e.g., umbilical and other herniæ. (2) Metabolic: gout, glycosuria. (3) Respiratory and cardiac troubles, bronchitis; cerebral hæmorrhage in plethoric type. Pneumonia and anæsthetics badly borne.

Diagnosis.—Usually simple. *In myxœdema*, skin dry and harsh.

Prognosis.—Expectation of life diminished by tendency to complications.

Treatment.—Marked obesity, to obtain any satisfactory result, must be treated strictly by scheme, with measured and weighed diet, and regular weighing of patient.

DIET—Scheme must regulate. (1) *Carbohydrates* practically excluded, except portion of bread not greater than 4 ounces. No sugar, potatoes, or puddings. (2) *Fats*: strictly limited, but butter in reasonable amount, being easily assimilated (e.g., 4 ounces weekly). (3) *Proteins*: amount increased, i.e., meat and fish. (4) *Fluid*: is usually reduced to unjustifiable extent: give 35 to 40 ounces daily: allow up to 50 ounces. No malt liquors, preferably no alcohol: best is claret or little whisky. Fluid should be reduced in cardiac cases and in those with much sweating.

CALORIC VALUE OF DIET.—1200 to 1500. One starvation day weekly, or the regular omission of one meal daily often effective.

SPECIAL ARTICLES.—Fruit allowed (not stewed, owing to sugar necessary). No rich sauces, appetisers, fat or rich meat. Soups best excluded. Vegetables allowed, except potatoes and peas.

BANTING'S DIET.—Mainly meat and fish: about 1 pound daily. No fat. Bread 2 to 3 ounces. Vegetables except potatoes. Fluids not greatly restricted, principally weak tea: some alcohol allowed.

May be well modified by allowing butter, somewhat more bread, and less meat, and reducing alcohol.

Other diets by Oertel (especially for "fatty hearts"), Von Noorden, etc.; but Banting's, modified to requirement, is simplest basis.

STRICT DIET for not more than six consecutive weeks. Loss of weight not to exceed 14 pounds a month.

MILDER FORMS.—Treated by further modifications on above bases, but always on definite scheme.

EXERCISE AND GENERAL HYGIENE.—*Exercise* regulated during strict diet: excessive exercise often attempted by subjects—always fails to reduce obesity, and often increases it, *unless diet is controlled*.

DRUGS.—*Thyroid extract* only constantly effective drug: *acts also on protein tissues*, and hence dangerous and inadvisable: *dose not exceeding* gr. v twice daily: give plentiful protein in diet: heart also must be carefully watched.

Other drugs in certain proprietary medicines are *Fucus vesiculosus* (bladder wrack): contains iodine and may increase thyroid secretion: efficiency unproved. *Citric acid*: no proof of efficiency.

IN CHILDREN.—Diminish carbohydrates and fat. Regular exercises. Results of treatment usually slight.

OTHER LIPOMATOSES.

1. **Adiposis Dolorosa** (*Dercum's disease*).—Four characteristics: (1) Obesity; (2) Pain and tenderness of fat; (3) Asthenia; (4) Psychical changes. Cause of pain unknown. Possibly lymphatic disturbance with slight degree of inflammation (cf. LIPOMATOSIS).

ETIOLOGY.—Females predominate: usually in middle age. Syphilis, alcoholism, and traumatism have been recorded.

MORBID ANATOMY.—Tumours of pituitary have been recorded in many typical cases.

SYMPTOMS.—Two types described: (1) *Localized form*; (2) *Diffuse form*. Obesity usually present previously, but rarely; the painful areas are the only deposits of fat.

LOCALIZED FORM.—Commonest. With previous obesity, there occurs *a painful local mass*, usually slightly raised and reddened, of another few inches, besides in a few days, leaving *diffuse painful nodule*. Recurs in other areas. After many weeks may cease, but the multiple painful nodules remain. No special nerve distribution. Other occasional symptoms: *a tacrine*, often marked local hyperæsthesia and anæsthesia; *but may be psychical changes*.

Distribution.—Back, neck, upper chest, arms and thighs. Face, hand, feet always secure.

DIFFUSE FORM.—Entire fat tender, but no local areas, nodules. Probably a different condition.

TREATMENT.—Palliative: Aspirin, etc., for pain. Thyroid and pituitary extracts have been tried.

2. **Lipoma.**—Local innocent encapsulated tumour of fatty tissue. Often multiple. May be painful.

3. **Diffuse Lipomatosis of Neck.**—Increase of fatty tissue around neck: may be enormous. Almost always in alcoholic males. May be local lipomata, general obesity, or sometimes wasting. Also termed 'adeno lipomatosis', as tissue may contain lymphatic glands.

4. **Dystrophia Adiposo-genitalis** (Fröhlich), (*Adiposis cerebralis*).—Due to abnormal pituitary secretion. (*See* PITUITARY GLAND.) *In cretinism and myxœdema*, local or general deposits occur of an abnormal fatty tissue.

CHAPTER LXIII.

HÆMOCHROMATOSIS.

(*Diabète bronzé.*)

A rare disease, probably due to an error of metabolism, and characterized by widespread pigmentation, by fibrosis of the liver and other organs, and usually by diabetes.

Etiology.—Age: 30 to 60 years. Almost unknown in women. No predisposing factors known.

Morbid Anatomy.—General pigmentation of tissues, brown or slaty colour.

LIVER.—Large, smooth, brick red. Histology: (1) Pigment very abundant in liver cells and fibrous tissue. (2) Multilobular cirrhosis.

SPLEEN.—Enlarged, pigmented, fibrotic.

PANCREAS.—Pigmented and fibrotic, either small or fatty.

LYMPHATIC GLANDS, HEART, AND INTESTINES also pigmented.

NATURE OF THE PIGMENT.—Two forms: (1) *Hæmosiderin*, iron containing pigment, in parenchyma of liver and other glands. (2) *Hæmofuscin*, iron free, in muscles of heart and intestines. Skin pigment is also iron free.

Pathogenesis.—*Pigment* is deposited in essential cells, which necrose, pigment thus set free in interstitial tissue causes fibrosis, whence cirrhosis of liver and also of pancreas, causing diabetes.

ORIGIN OF PIGMENT.—*An error of metabolism, by which pigment is formed from protein* (Sprunt). Rejected theories include (*a*) Increased hæmolysis and formation of blood pigment. Against this is absence of jaundice, anæmia, and hyperplasia of bone marrow. (*b*) Suprarenal disease: no evidence of existence.

Symptoms.—(1) *Pigmentation*: Brown to slate colour, mainly on exposed parts. (2) *Enlarged liver and spleen*: Smooth and uniform. (3) *Diabetes*: Occurs in 80 per cent., late in disease but severe and rapid.

Two groups distinguished: (*a*) With diabetes (*diabète bronzé*). (*b*) Without diabetes. *Ascites and other signs of hepatic cirrhosis* develop in either.

Prognosis.—Bad. In diabetic group, death usually in coma within one year of glycosuria. If no glycosuria, progress may be slow.

Diagnosis.—From: (1) *Addison's disease*, by glycosuria and enlarged liver and spleen. (2) *Hypertrophic biliary cirrhosis*, by absence of jaundice and presence of glycosuria. (3) *Splenic anæmia*. In this disease spleen is very large, and anæmia advanced; no pigmentation.

Characteristics.—(1) Pigmentation of skin (sometimes slight). (2) Enlarged liver; and (3) Glycosuria.

Treatment.—As in diabetes and cirrhosis of liver.

Section IV —**Diseases of Metabolism and Diseases of Deficiency,** *contd.*

B. DISEASES OF DEFICIENCY.

CHAPTER LXIV.

ACCESSORY FOOD FACTORS: VITAMINS.*

Natural foods contain certain constituents present in minute amounts; but if these be removed, such foods are wholly unable to support nutrition, and symptoms of actual disease may develop. Owing to their small amount these bodies must be unconnected with the supply of energy and protein, yet they are necessary for complete normal metabolism. Food supply therefore can no longer be estimated merely in terms of the four fundamental units, protein, carbohydrate, fat, and inorganic material.

These substances are now known as 'accessory food factors' or vitamins. They are present in all natural diets of men and animals and are present in sufficient supply in food so long as it is reasonably varied, has not been separated into parts artificially or accidentally, and has not been exposed to any destructive process. They are apparently formed only in the tissues of plants, and cannot be synthesized by animals. They have not been isolated.

VITAMINS at present recognized are:

1. *Fat soluble A.* Present in fats necessary for growth. This or a closely similar vitamin is one of the factors in rickets.
2. *Water-soluble B.* Present especially in seeds and eggs, necessary for growth, and probably identical with the vitamin concerned in beri-beri.
3. *Anti-scorbutic Vitamin* or *Water soluble* C_2—Present especially in fresh vegetables and fruits, and necessary for the prevention of scurvy.

Other vitamins may exist, e.g., for pellagra.

DISCOVERY OF VITAMINS.—Experiments with purified diets.

LUNIN, 1881.—Animals died in one month on artificial purified diet containing the supposed essential ingredients of milk, viz., caseinogen, milk fat, milk sugar, and ash of milk.

At the time this was attributed to: (1) Monotony of diet. (But pure milk will sustain life.) (2) Lack of flavouring, and hence loss of appetite.

Beri-beri had been largely elucidated before the following researches were published.

OSBORNE AND MENDEL, 1911.—Rats died on diets composed of isolated food substances, viz., starch, sugar (lactose), lard, inorganic salts, with agar as a basis.

* Report of Joint Committee of Lister Institute and Medical Research Committee.

Vitamins—Discovery of, *continued*

STEPP, 1911–12.—Mice died on diets, otherwise satisfactory, but extracted with alcohol and ether, but could be saved by addition of these extracts

HOPKINS, 1912.—Young rats died rapidly on a diet of purified food substances, but lived and grew on addition of milk (4 per cent of diet), milk extracts, or yeast. He concluded that some 'accessory food factors' were essential for growth in young animals, as he had suggested previously in 1906

OSBORNE AND MENDEL, 1913.—Discovered that the active substance was concentrated in the butter fat fraction of milk. Also was found to accompany the fat when extracted with ether

Until the following research, this was believed to be the only accessory substance necessary to supplement a diet of purified constituents in order to produce growth

McCOLLUM AND DAVIS, 1915.—Study of rice diets proved the existence of a second accessory factor essential for normal nutrition during growth. They named the two factors now known (1) *Fat soluble A*, (2) *Water-soluble B*

Also proved that *Water soluble B* is present in milk and separated with difficulty from milk sugar; this explains its previous escape from observation since it was present in lactose used as sugar in many experimental diets

Fat-soluble A.—Present especially in (a) green leaves, (b) embryos of many seeds. In seeds is probably in loose combination with some substance not fat, since (1) Simple processes for extracting fats do not remove it: pressure extraction with solvents. Hence absent from vegetable fats thus prepared. (2) If finely divided seed embryos be treated with alcohol, the combination is destroyed and *A* can then be removed by ether

Animal tissues normally have considerable stores of *A* in reserve

PROPERTIES.—

1. Soluble in fat solvents, e.g. ether
2. Insoluble in water
3. Comparatively resistant to heat, but gradually destroyed at 100° C. in about 4 hours
4. Destroyed in hardening oils by action of hydrogen, method widely used in preparation of edible fats
5. Stable to alkalis in conditions employed in hydrolysis of fats

DISTRIBUTION IN FOOD SUBSTANCES.—

PRESENT IN (1) Fats: milk, butter, cream, egg yolk, dried eggs. Cod liver oil and many animal fats and oils. Cheese, if from whole milk. (2) Vegetables: cabbages and most vegetables; potatoes, in sufficient extent. (3) Cereals and pulses: pulses (peas, beans, etc.), *embryos* of cereals. (4) Meat and fish: liver, kidney, heart muscle; fat fish, e.g., herring, salmon; presence in lean meat doubtful.

ABSENT FROM: (1) Vegetable oils, e.g., olive oil, cotton-seed oil, linseed oil. (2) Lard. (3) Yeast. (4) Malt extract. (5) Meat extracts.

In accordance with above, absent from: white bread; salad and frying oils; margarine, except when prepared from animal fat (other than lard); custard powders and egg substitutes. Deficient in most patent and proprietary foods.

EFFECTS OF ABSENCE OF *FAT-SOLUBLE A* FROM THE DIET —

ADULTS.—Able to live for long periods in its absence. General health suffers.

GROWING ANIMALS.—Young rats grow for a short period, then growth commences to cease, weight becomes stationary, and may fall, but death more commonly from septic complications; special tendency to inflammations of the eye, as xerophthalmia.

PHYSIOLOGICAL ACTION may be: (a) Concerned in metabolism of fats. (b) Concerned with general nutrition of cells. Latter hypothesis supported by: (1) Little loss of body fat. (2) No signs of abnormal fat metabolism. (3) Growing animals partially affected.

RICKETS probably results from a deficiency of this vitamin.

Water-soluble B. Present especially in [illegible] and eggs of animals. Easily extracted.

Animal tissues normally contain no reserves of *B*.

PROPERTIES

1. Soluble in water. Also in alcohol but not in the other fat solvents.
2. Moderately resistant to heat. Survives 150° C. for 1 to 2 hours, but destroyed at 120° C. in half an hour.
3. Very resistant to drying.

DISTRIBUTION IN FOOD SUBSTANCES. Especially present in seeds and eggs of animals.

PRESENT IN: (1) Cereals and pulses. In cereals is contained mainly in the embryo, which is removed in machine milling: hence polished rice and white flour contain no vitamin. In pulses is distributed through at seed. (2) Eggs, fresh and dried. (3) Yeast. (4) Vegetables. Potatoes (amount sufficient, but not very large). (5) Meat (in moderate degree), liver, kidney. (6) Milk, amount slight.

ABSENT FROM: Fish. Fats of all kinds. Meat extracts.

In accordance with above, absent from: white bread; polished rice, tinned and autoclaved foods.

EFFECTS OF ABSENCE OF *WATER-SOLUBLE B* FROM THE DIET —

GROWING ANIMALS.—Growth ceases almost at once, and rapid loss of weight follows.

PERIPHERAL NEURITIS develops in animals of any age.

BERI-BERI is caused by the absence of vitamin which is

Vitamins—Water-soluble B, *continued.*

probably identical with ***Water-soluble B***. This is supported by the similarity of the distribution of the two vitamins and of the symptoms which result from their absence.

EXPERIMENTAL BERI-BERI (*Avian polyneuritis*).—Fowls and pigeons are very susceptible to beri-beri diet and absence of ***Water-soluble B***. Onset of symptoms in 15 to 25 days: death in 24 to 48 hours in absence of treatment. Symptoms: weakness of legs, wing-drop, head retraction, general paralysis. Cured by administration of vitamins, e.g., yeast, alcoholic extract of rice polishings (by mouth or by injection). Recovery on treatment is extraordinarily rapid. A bird *in extremis* may be flying about within 2 hours of an injection, apparently in perfect health.

Anti-scorbutic Vitamin: Water-soluble C.—Scurvy was recognized in the 17th century as due to long deprivation from fresh foodstuffs, and its prevention and rapid cure by fresh vegetables and fruit juice were known.

EXPERIMENTAL SCURVY

Axel Holst, in 1907, found that guinea pigs on a scurvy diet rapidly developed symptoms similar to human scurvy, and now accepted as identical.

At the Lister Institute these experiments have been continued, and have proved that scurvy is due to absence of a vitamin which is neither *Fat soluble A* nor *Water-soluble B*.

PROPERTIES.—

1. Very sensitive to drying, and rapidly destroyed thereby.
2. Very sensitive to heat. Temperature of 60° C. for one hour destroys 80 per cent. The rate of destruction does not increase very rapidly with further rises of temperature.
3. Rapidly destroyed by alkalis.
4. Protected by acids.
5. Soluble in water and in alcohol.

DISTRIBUTION IN FOOD SUBSTANCES.—Present in plant tissues in which active metabolism is taking place.

PRESENT IN: (1) Fresh vegetables, especially cabbages (raw or cooked), onions, juice of swedes, authentic in potatoes (cooked). (2) Fruit juice, especially of oranges and lemons (fresh or preserved juice). (3) Raw meat juice, milk, and certain dried fruits to a moderate extent.

ABSENT FROM: Dried vegetables, dry cereals and pulses, tinned and autoclaved foods; yeast.

GERMINATED PULSES AND CEREALS.—Though absent from dry pulses and cereals, the vitamin *appears* in these if soaked in water and allowed to germinate for a few days.

'LIME JUICE'.—Formerly was known to cure or prevent scurvy, but evidence has accumulated of its failure to do so in recent Polar and similar expeditions. This failure has led in recent decades to other theories for scurvy, such as tainted meat. Experiment proves that preserved lime juice contains no anti-scorbutic vitamin. The explanation is that until 1850 'lime juice' was really prepared from Mediterranean

lemons. Since then West Indian limes have been used, the preserved juice of which is not anti-scorbutic. Investigation shows that since this date lime juice has never prevented or cured scurvy (Henderson Smith).

Period of Development of Diseases of Deficiency.—

BERI BERI.—80 to 90 days.

SHIP BERI BERI.—Probably more than one vitamin is absent: develops more rapidly.

SCURVY.—Minimum is 4 months. More commonly nearly 8 months.

INFANTILE SCURVY.—In agreement with above, appears at age of 6 to 8 months.

Diets of Infants.—*Application of knowledge of vitamins.*

1. BREAST FEEDING.—Mother should take food rich in vitamins, especially in *Fat-soluble A*, viz. milk, eggs, butter. Margarine only of value if prepared from animal fats (other than lard).
2. ARTIFICIAL FEEDING.—

COW'S MILK.—Contains all vitamins, but is not a powerful anti-scorbutic.

DRIED MILK.—Contains sufficient *A* and *B*, but is not anti-scorbutic.

CONDENSED MILK.—Contains vitamins, but in sweetened forms the dilution necessary is often so great as to reduce the amount received by infants below what is essential.

PATENT AND PROPRIETARY FOODS.—Majority are seriously deficient in fat and in *Fat-soluble A*, probably many also in *Water-soluble B*, all deficient in anti-scorbutic vitamin.

BOILED AND PASTEURIZED MILK.—Deficiency in anti-scorbutic factor probably varies with method adopted, certainly deficient in many cases.

HUMANIZED MILK.—Cow's milk is modified to approach the composition of human milk, usually by addition of milk products, and in such cases contains the necessary vitamins.

An additional anti-scorbutic should be given to all infants who are reared on any artificial food other than raw cow's milk, and may well be given to all infants, even breast-fed. Orange juice is the best.

CHAPTER XXV.

BERI-BERI.*

(*Kakké. Endemic Multiple Neuritis.*)

A condition due to the absence from the diet of certain substances (vitamins), and characterized by multiple peripheral neuritis, and by œdema, effusions, and cardiac weakness.

Beri-beri is the prototype, the first established and most fully investigated of 'diseases of deficiency', and hence of great importance.

* Main details from Vedder's monograph, *Beri-beri*.

Beri-beri, *continued.*

Distribution.—In all regions in which rice is the staple diet, and in certain other places and circumstances from local causes In Japan, parts of China and India, Dutch East Indies, the Philippine Islands, and Malay, affects large numbers

Etiology.—

A 'disease of deficiency' due to absence of accessory food factors or vitamins. The vitamin is probably identical with *Water soluble B* (*see* p 339).

An association of beri beri with rice dietary was early noted by many observers, but for long was not generally accepted Some important steps in the brilliant work elucidating this disease are —

TAKAKI in 1884 eradicated the disease from Japanese Navy (from 30 to 40 per cent to under 1 per 1000) by adding meat and milk to rice diet. His explanation was obviously erroneous viz, deficiency of nitrogen according to Voit's Standard Consequently in spite of success his views and measures were generally unaccepted

EIJKMANN 1893, produced *P. nervosum* [illegible] in fowls on diet of cooked rice and asserted identity with human beri beri

VORDERMAN, 1895, proved experimentally in Java prisons that disease was produced by polished rice, and cured or averted by unpolished rice This attracted no attention

GRIJNS, 1901 repeated Eijkmann's experiments and proved that a bean Katjang idjo (*Phaseolus radiatus*) was protective and curative for fowls on polished rice diet

HULSHOFF POL 1901 1904 applied this to human beings and found 150 grm of the bean daily to be equally protective and partially curative

BRADDON 1907, in monograph on beri beri showed that disease followed use of polished rice and was prevented by cured or parboiled rice This was first recorded proof that condition was due to rice Ascribed to [illegible] developing in rice deprived of pericarp

SCHAUMANN ascribed disease to removal of phosphates in aleurone layer but phosphorus inorganic or organic failed to cure or avert condition

FRASER AND STANTON 1907 1908 proved etiology definitely

1. Two parties of coolies fed the one on polished and the other on unpolished rice then vice versa Beri beri always occurred in party on polished rice, and was cured by unpolished rice

(2) Beri beri proved to be a 'disease of deficiency' by following feeding experiments on fowls (a) Polished rice after extraction with alcohol beri beri resulted The extraction with alcohol was an attempt to remove toxins (b) Unpolished rice after extraction with alcohol beri beri resulted. Decisive experiment originally performed as control to (a) (c) Polished rice with added extract of unpolished rice no beri beri (d) Undermilled or unpolished rice: no beri beri.

Therefore milling removes some essential substance from the rice, and beri-beri is a 'disease of deficiency'

THE RICE GRAIN AND EFFECTS OF MILLING.—A grain consists of (i) Pericarp or thin outer layer, (ii) Aleurone layer, containing all the *phosphates and fats*, (iii) Germ or embryo, (iv) Endosperm, the bulk of the grain, consists of starch granules. In 'polishing' or steam milling, practically all the aleurone layer is removed. This was supposed to contain the anti-beri beri substance. Recently Chick and Hume have proved the presence of this substance mainly in the germ, which is simultaneously removed.

OCCURRENCE AND DISTRIBUTION OF BERI BERI.—Explained by properties and distribution of vitamin. Apart from polished rice diets outbreaks will occur anywhere if diet is: (a) Deficient in anti beri beri vitamins, (b) Sterilized or auto fayed. This explains many outbreaks previously used as arguments against rice theories, e.g. amongst Norwegian fishermen in ships, jails and institutions in many lands.

Morbid Anatomy.—Main changes correspond to the three groups of symptoms.

1. NERVOUS SYSTEM.—Peripheral nerves: changes characteristic of peripheral neuritis, vagus and phrenic affected. Spinal cord: in chronic cases degeneration of posterior columns, posterior spinal ganglia, and anterior horn cells.
2. EFFUSIONS.—Hydropericardium (up to 3 pints) in 60 per cent of fatal cases. Œdema of lungs, pleural and other effusions common. General anasarca in wet type.
3. HEART.—*Hypertrophy of right ventricle with little change in left characteristic.* Myocardial degeneration.

LIVER, KIDNEYS, SPLEEN: no compression changes, slight.

Symptoms.—

PERIOD OF DEVELOPMENT: Varies, 10 days usually elapses on a beri beri diet before appearance of symptoms.

THREE MAIN GROUPS.—(1) Multiple peripheral neuritis, (2) Œdema and effusions, (3) Cardiac weakness.

FOUR CLINICAL TYPES are recognized, with varying degrees and combinations of these groups.

(1.) RUDIMENTARY OR LARVAL BERI BERI.—

Onset: Gradual. All typical symptoms often present, but to slight degree, viz. weakness of legs and paræsthesia, slight œdema of legs and face, palpitations, pain, and cardiac weakness.

Progress: May disappear, remain stationary for years, or progress into the other clinical types, which commonly commence thus.

This type is in reality *mild chronic beri beri.*

(2.) DRY OR ATROPHIC BERI BERI.

Onset as above. Then paralysis is rapid and widespread, wasting and atrophy severe, with anæsthesia and paræsthesia. No œdema. Cardiac symptoms slight. Becomes helpless.

Beri-beri—Symptoms, *continued.*

(3) 'WET' OR DROPSICAL BERI-BERI.—
Onset as first type. Then rapid œdema, anasarca, and effusions: cardiac symptoms and dyspnœa marked. Paralysis slight: atrophy often noted as œdema clears.

(4) ACUTE PERNICIOUS BERI-BERI OR CARDIAC FORM.—
Onset as in first type. Then acute cardiac failure, with pain, palpitations, and dyspnœa. Paralysis often severe. Rare form, but high mortality. Duration, few days to several weeks.

Special Features of Symptoms.—
PERIPHERAL NEURITIS*—Both motor and sensory fibres affected. Lower extremities first attacked. In severe cases widespread. Sphincters escape. Atrophy rapid. No ataxia. Knee-jerks absent early.
Sensory—(i) Anæsthesia: constant, all sensations affected, commences over tibiæ (an early and simple test). (ii) Paræsthesia common. Never hyperæsthesia. (iii) Muscles, especially calf, very tender.

ŒDEMA AND EFFUSIONS—*Œdema* commences in feet and spreads upwards in 'wet' type, extreme and generalized. Also in 'wet' type *œdema of lungs*, common cause of death; *hydrothorax*, often large; œdema of meninges. *Hydropericardium*: present in most fatal cases of all types; rarely exceeds 8 ounces.

CARDIAC SYMPTOMS—(i) *Palpitations*, rarely absent. (ii) Hypertrophy and dilatation of *right heart*. Pulse usually rapid.

OTHER SYMPTOMS—*Blood*: anæmia not marked. (In tropics, blood picture often complicated by presence of parasites.) *Urine*: no albuminuria. *Spleen*: not enlarged. *Temperature*: normal. *Digestive system*: appetite normal; constipation common; vomiting rare but serious. *Mental condition*: unaffected. No tremors.

Note on Clinical Types. These are closely allied, and any one may follow the others, e.g., 'wet' type in a previous 'dry' beri-beri. Possibly may be slightly different vitamins concerned. Types may co-exist, and muscular atrophy and weakness become apparent as œdema improves in 'wet' type.

Prognosis.—Serious in acute cardiac type. With treatment good in other forms. The peripheral neuritis improves but slowly (as from other causes). Mortality very low if nutritious anti-beri-beri diet available.

Diagnosis.—Simple in a beri-beri country. In non-rice beri-beri, is often unsuspected. Diagnosis from—
NEPHRITIS—No albuminuria.
HEART DISEASE (often difficult): No murmurs. Anæsthesia over tibiæ. Occurrence of numerous cases.

* When asked to give the causes of multiple neuritis, a student should refrain from mentioning beri-beri first. This reply often has an acute irritant action on an examiner. 'Alcohol' is preferable.

ALCOHOLIC NEURITIS.—No nervous or mental disturbances.
MALARIAL NEURITIS (rare).—No fever. No splenomegaly.
Rarely from: trichiniasis, ankylostomiasis.

Prophylaxis.—Dietetic: a mixed nutritious diet. If this is not available, the addition of substances rich in the vitamin: yeast, eggs, peas and beans, or fresh meat or undermilled rice.

Treatment.—Dietetic, as above. Symptomatic also for the peripheral neuritis and in the acute cardiac types. Milk is valuable adjuvant to diet. In severe cases give rice polishings, 150 grm. daily, or their alcoholic extract, or pure yeast.

ATYPICAL FORMS AND CONDITIONS ALLIED TO BERI-BERI.

The discovery of beri-beri 'vitamin' and its properties has enabled these to be more properly understood.

1. **Ship Beri-beri.**—Onset gradual. Symptoms: (1) Œdema of ankles, spreading upwards; (2) Tingling and weakness in legs; (3) Rapid pulse. *No hæmorrhages or spongy gums. No albuminuria.* Neuritis rarely severe, and acute cardiac form rare. On change of diet, recovery rapid. Diet often mixed, and of high nutritional value; defect solely in vitamins, often due to sterilization of food. Became frequent among Norwegian fishermen after 1894, following change by law from a poor but vitamin rich diet to one superior but vitamin free. Period of development shorter than rice beri-beri (90 days), but is same disease. Scurvy may co-exist.
2. **In Asylums, etc.**—Similar to ship beri-beri in etiology.
3. **Epidemic Dropsy.**—Condition described in Calcutta and Mauritius. Distinction from beri-beri formerly claimed owing to occasional rashes, mild pyrexia, and slightness of neuritis.
4. **War Œdema.**—Nerve and heart symptoms slight or absent. No albuminuria. McCarrison's theory: (1) Inanition results in hypertrophy of adrenal and hyperadrenalinæmia; (2) Hyperadrenalinæmia raises the intra-capillary pressure; (3) Œdema results.
5. **Infantile Beri-beri.** Causes enormous infantile mortality. McLaughlin and Andrews, 1909, found changes at autopsy identical with adult beri-beri. Chamberlain and Vedder, 1912, cured cases by rice polishings.

SYMPTOMS.—(1) *Acute type.* Common. Sudden paroxysmal pain and tachycardia. Repeated attacks. Death within few hours. (2) *Chronic type.* Less common. Vomiting, constipation, tachycardia and œdema. No paralysis. Death as in acute type.

AGE.—One to three months, never over a year. Always breast fed infants of beri-beri mothers, and due to deficient vitamins in the milk.

TREATMENT (Chamberlain and Vedder).—Give 20 drops of extract of rice polishings two hourly, and later rice polishings to infant and mother. Many recoveries.

CHAPTER LXVI.

RICKETS.

(*Rachitis.*)

A disease of metabolism occurring in late infancy, due to inappropriate diet, and characterized mainly by changes in the bones

Etiology.—

AGE —Most commonly observed at one to two years rare under six months and over three years. Very rare in breast fed infants

Sexes equal. Heredity absent Syphilis aggravates, but does not originate. Prevalent in poorer classes, city dwellers and temperate regions. Occurrence of congenital rickets is doubtful formerly confused with achondroplasia

RICKETS CONSIDERED AS A DISEASE OF DEFICIENCY

Rickets in babies occurs on diet deficient in fats, and is cured or alleviated by supplying such deficiency Researches by E Mellanby and others support the following propositions —

1. Puppies develop rickets on a diet deficient in a vitamin identical with or similar to *Fat soluble A* (antiraphitic vitamin)
2. Absence of such vitamin is not sole cause of rickets sufficient absence of *Fat soluble A* inhibits growth while rickets develops most in rapidly growing puppies and in plump infants More than one factor is thus involved in rickets
3. Excess of carbohydrates (e g bread) in diet is probably a factor

CONCLUSION A diet containing an excess of carbohydrates (and possibly also other foods) selected especially when associated with rapid growth and with deposition of fat requires a certain amount of antirachitic vitamin and rickets results in absence of this amount —i e an absolute or relative vitamin deficiency

OTHER THEORIES OF RICKETS —

INFLUENCE OF ENVIRONMENT Certain authorities believe that rickets results from unhygienic surroundings and deny influence of vitamins Evidence however leads to conclusion that errors in environment will not produce rickets if the diet be adequate

DEFICIENCY OF FAT —Is not the essential factor since rickets may develop on a diet containing much fat if of vegetable origin e g, linseed oil or, to a less extent, cotton seed oil

DEFICIENCY OF CALCIUM —Administration of calcium will not prevent rickets if the diet be otherwise deficient Deficiency of calcium will not produce rickets if the diet be adequate but will cause bones to be soft and thus aggravate rickets

Morbid Anatomy.—

DEVELOPMENT OF NORMAL BONE —(A) *Intracartilaginous or endochondral ossification* (epiphyses) Characterized by

orderliness, and by confinement and completion of each process in a definite zone. Zones are: (i) *Normal cartilage* Blood-vessels absent or very scanty (ii) *Zone of proliferation of cartilage* Cartilage cells enlarge, multiply, and become arranged in parallel columns Blue tint. (iii) *Zone of primary calcification* Matrix becomes *calcified* between columns of cells. Forms a yellow line of not greater breadth than $\frac{1}{16}$ inch. (iv) *Region of ossification* Capillary loops end on sharp line just short of previous zone Processes occurring on the walls of the spaces containing the loops are: (a) Large osteoclasts absorb the calcified matrix; and then (b) Osteoblasts *deposit true bone* in the remnants of matrix By this method, bones increase in length. (v) *Intramembranous ossification or subperiosteal bone formation* By growth of capillary loops, and by osteoblasts depositing bone in connective tissue below periosteum Bones thus increase in width]

In rickets, bone development is characterized by disorderliness and lack of proportion of various processes

INTRACARTILAGINOUS OSSIFICATION (EPIPHYSES)—

1. *Multiplication of cartilage cells excessive* and no arrangement in columns, a broad blue area results Hence *enlargement of epiphyses*
2. *Calcification deficient.*
3. *Excessive vascularity* Capillary loops invade zones of calcification, proliferation, and even normal cartilage.
4. *Formation of true bone defective* (a) Osteoclasts absorb too much calcified matrix, and (b) Osteoblasts deposit not true bone but soft *'osteoid tissue'* deficient in lime salts Hence *softness of bone*

A transverse section through epiphysis will thus show in a single field irregular proliferated cartilage cells, attempts at column formations areas of partial and irregular calcification, capillary loops areas of osteoid tissue, and may be areas of fully formed true bone

PERIOSTEUM—Vascular layer much thicker than normal, also distribution irregular Increases size of epiphyses

CHANGES IN THE BONE MARROW—Red cells very numerous Myelocytes diminished

LIME SALTS IN RICKETY BONES—From 30 to 50 per cent of weight, compared to 60 to 65 in normal bone As *rickety condition improves*, deposit of lime salts becomes excessive, possibly from great vascularity; hence bone finally is harder and more brittle than normal, and deformities become permanent

Symptoms.—Rickets is a disease of nutrition, of which the bone changes are only one, though the most important, manifestation

GENERAL DESCRIPTION.—Onset insidious (i) Age, most commonly in second year; (ii) Diet deficient in fat, (iii) Child plump but flabby, irritable, (iv) Delay in sitting up and walking or 'goes off his legs'; (v) Profuse sweats, (vi) Dentition delayed, (vii) Tendency to bronchitis, diarrhœa, and in severer cases, convulsions. *On examination*: (i) Head large, anterior fontanelle patent, frontal bossing, (ii) 'Rachitic rosary', pigeon

Rickets—Symptoms, *continued.*

breast, and Harrison's sulcus; (iii) Epiphyses enlarged; (iv) Deformities of long bones; (v) 'Pot-belly' and palpable liver; (vi) Laxity of ligaments.

PRINCIPAL SYMPTOMS.—

GENERAL NUTRITION.—Often fat, but flabby. In severe forms, wasting. Irritable. Poor appetite.

SWEATING.—Early and constant. Especially at night and of head.

BRONCHITIS.—Common, also bronchopneumonia; serious.

ALIMENTARY SYSTEM.—(1) Abdomen distended, 'pot bellied', flatulence, muscular relaxation, and descent and enlargement of liver; (2) Enteritis and intestinal disturbances common. (3) Liver frequently palpable, spleen less often, due to displacement more than enlargement.

DENTITION DELAYED.—Very constant. Often no teeth at 12 months. Caries early.

BONES AND LIGAMENTS.—*See below.*

NERVOUS SYSTEM.—Convulsions: rickets is a very common cause. Laryngismus stridulus and tetany usually associated with rickets. *No mental changes.*

TEMPERATURE.—Normal, unless complications.

BLOOD.—Variable degree of anæmia.

Changes in the Bones and Ligaments.—

THORAX.—*Beading of the ribs* or 'rickety rosary', enlarged epiphyses at costochondral junction, most constant and often earliest symptom of rickets. Subsequently diminishes, rarely recognizable at puberty. *Pigeon breast*: sternum projects, especially lower half, section through thorax becomes triangular, costochondral junction sunk. *Harrison's sulcus*: groove from ensiform cartilage outwards, with costal margin curved upwards. (These latter two changes also occur in conditions with inspiratory obstruction, e.g., enlarged tonsils.)

EPIPHYSES.—Enlarged. Especially lower end of *radius*, and frequently of tibia and femur.

HEAD.—

1. *Enlarged*, square and flattened on vertex ('caput quadratum'). Less commonly, lengthened antero-posteriorly.
2. *Anterior fontanelle patent* until 2 or 3 years (normally closed at 1½ years).
3. Bossing of frontal eminences. 'hot cross bun' head.
4. Craniotabes. Not common. When present, frequently, but not always, co-existent syphilis.

CURVATURE OF LIMBS AND DEFORMITIES.—The long bones, being soft, bend from weight of body and muscular traction.

TIBIA.—(i) *Commonest deformity*: Curve at lower third, concavity on external surface; or a sharp forward bend. (ii) Curve at upper third, concavity on internal surface, viz., 'bow legs.'

FEMUR.—Less common. (i) General antero-posterior curve. Rarely, (ii) Coxa vara. (iii) Genu valgum.

SPINE.—Kyphosis, if child sits up.

OTHER DEFORMITIES AND RESULTS.—

Upper extremity: curvatures uncommon, unless child crawls on arms.

Pelvis: flattened antero-posteriorly, may be small and contracted: important in females

Permanent dwarfing may result from deformities and disease of epiphyses

Fractures, common, especially 'green-stick'.

LIGAMENTS.—*Extreme laxity*. This and muscular weakness aid deformities.

MUSCLES.—May be great weakness, even suggesting paralysis.

Course and Prognosis.—Rickets reacts readily to correct treatment: active process controlled in about six months. Recrudescence very rare

MORTALITY.—Results mainly from complications of *bronchitis and diarrhœa* Rarely from convulsions, laryngismus stridulus, and tetany Direct mortality very slight

DEFORMITIES.—Improve, and if slight may disappear with rest, treatment, and in subsequent growth If severe, many persist, e.g., *rickety pelvis*, *'bow legs'*.

BONES.—Subsequently harder than normal, and more brittle.

COMPLICATIONS (i) Respiratory, (ii) Alimentary disturbances. Syphilis increases severity of rickets In splenic anæmia of infants, rickets often present

Diagnosis.—In mild types, difficult: in severe, simple *Diagnosis* occasionally must be made from (1) Infantile paralysis sudden onset, reflexes absent (2) Spinal caries sharp local curve. (3) Hydrocephalus.

Prophylaxis.—

Breast-feeding 8 to 9 months Rickets subsequently very rare

Sufficiency of fat for artificially fed children. With diluted cow's milk, add cream Avoid starchy farinaceous foods.

In second year of life, child should have 1½ to 2 pints of milk daily.

Treatment.—

DIET.—Essential. (i) Sufficiency of fat. To each bottle of diluted milk, add teaspoonful of cream Give cod-liver oil ʒss to ʒi. t.d.s., combines well with malt extract. (ii) Avoid excess of starch, i.e., bread, potatoes (very frequent error in second year) Yolk of egg (one daily) (*See* VITAMINS, 'Diets of Infants', p 341.)

REST.—To prevent and correct deformities. Especially if fat. With severe types, 3 months' complete rest. Long splints beyond feet will successfully prevent child walking.

Rickets—Treatment, *continued.*

GENERAL HYGIENE.—Fresh air. Warm clothing. Bath daily, with cold douching. Treat bronchitis and digestive disturbances (gray powder useful).

DRUGS.—Tonics: syrup. ferri phosphatis (over 1 year). Calcium salts are valueless. Influence of phosphorus unproved.

DEFORMITIES.—May need operative treatment later.

LATE OR ADOLESCENT RICKETS.

True rickets commences before 3 years of age. From 3 years and up to the age of 14 or 15 years conditions occasionally arise in which changes at epiphyses and development of bony deformities more or less closely resemble rickets. There are several groups, all rare at least in advanced degrees. Certain, but not all, are fairly directly connected with deficient fat, either in diet or by absorption. Some cases diagnosed as Still's disease probably belong here.

Provisional Groups.—

1. RECRUDESCENT RICKETS.—In some instances ordinary rickets has occurred previously, with recrudescence about age of 9 years.
2. RENAL INFANTILISM (*Renal Dwarfism*). See *Vol. 1.*
3. COELIAC DISEASE.—Late rickets may occur (Miller), obviously connected with deficient absorption of fat.
4. DEFICIENCY OF FAT IN DIET. Recorded as result of war condition.
5. WITH SPLENIC ENLARGEMENT AND BLOOD CHANGES RESEMBLING VON JAKSCH'S OR PERNICIOUS ANÆMIA.

Syphilis is certainly not a usual factor.

Renal Infantilism.* —Changes at epiphyses and deformities resembling rickets, with retarded physical development associated with chronic interstitial nephritis.

ETIOLOGY.—Sexes equal. No previous rickets or parenchymatous nephritis. No evidence of syphilis as a factor.

MORBID ANATOMY.—Kidneys very small; capsule not adherent and surface only slightly granular; interstitial tissue markedly increased, but slight changes in parenchyma; small vessels thickened. No obvious change in cardiovascular system or endocrine organs.

SYMPTOMS.—

ONSET.—Probably in infancy; patient usually small from birth; special symptoms and deficient growth noted at age of 6 years or later. Thirst and polyuria may be early and marked.

DEFORMITIES AND ABNORMALITIES OF JOINTS. Swelling at joints. Knees and wrists most affected; also ankles and costochondral junctions. No bossing of skull. Genu valgum often develops rapidly in second decade.

* See Barber, "Renal Dwarfism", *Quarterly Journal of Medicine*, 1911, April.

URINE.—Amount variable; may be very large, with low specific gravity. Albumin present: amount small, about 1 part in 1000: may be absent. Few hyaline casts.

CARDIOVASCULAR CHANGES.—Usually no obvious cardiac hypertrophy, thickening of peripheral arteries, or retinal changes: also normal blood-pressure (increased in some cases). Blood normal.

PHYSICAL DEVELOPMENT.—Growth retarded. Thin. Sexual infantilism in some, but not all. Intelligence normal.

X RAYS.—Changes resemble rickets.

PROGNOSIS.—Usually fatal uræmia in second decade.

CHAPTER LXVII.

SCURVY.

(*Scorbutus*.)

A 'disease' [illegible] 'new' due to the absence from the diet of certain substances (vitamins) contained in fresh food, and characterized by swelling of the gums, by hæmorrhages into the skin and subcutaneous tissues and from mucous membranes, and by anæmia.

Anti-scorbutic Vitamin.—See Chapter LXIV, p. 340.

Morbid Anatomy. Changes mainly due to hæmorrhages. Effusions large or small into skin, deeper tissues or subperiosteum. Petechiæ on pleura or pericardium. Infarcts in lungs and spleen. Gums swollen. Other organs, little change. Blood vessels, structure normal.

Symptoms.—

PERIOD OF DEVELOPMENT.—At least 4 months on scurvy diet; more commonly, nearly 8 months.

ONSET.—Insidious: general weakness, pallor, then symptoms of anæmia. Bruises very easily.

CHARACTERISTICS.

1. SWELLING OF GUMS.—Becomes extreme. Gums bleed easily, fœtor; teeth fall out. (Rarely, gums unaffected.)
2. HÆMORRHAGES.—(i) Hæmorrhages from mucous membranes, nose and mouth and subconjunctivæ, *but hæmoptysis, hæmatemesis, and hæmaturia rare.* (ii) Petechiæ or ecchymoses into skin and subcutaneous tissues, also intramuscular tissues or under periosteum; common in loose folds of skin, e.g., bend of elbow. Result from slight bruises. (iii) Deep hæmorrhages, often large, tender, pit on pressure; skin on surface red and hot. Ulceration common.
3. ANÆMIA.—Palpitations, often severe. Some œdema of the ankles, but no general anasarca. *Blood*: secondary anæmia, no leucocytosis.

Scurvy—Symptoms, *continued.*

Albuminuria usual. Temperature normal, unless complications. Constipation most frequent, but diarrhœa not uncommon. Alimentary system unaffected except anorexia from condition of gums.

Complications.—

GANGRENE OF LUNGS.—From septic inhalation, or from hæmorrhages in lungs.

NIGHT BLINDNESS.—Frequently associated. Relation doubtful

BERI-BERI.—May co-exist: usually preceding scurvy.

Diagnosis.—Simple when many cases. In sporadic cases, from: (1) Acute leukæmia, myeloid or lymphoid: blood examination. (2) Purpura hæmorrhagica: gums unaffected. Rarely, when gums unaffected, from various purpuric affections: by reaction to treatment.

Prognosis.—No improvement unless anti-scorbutic food provided Death from cardiac failure, or intercurrent disease, e.g., diarrhœa Has caused enormous mortalities among crews Reacts readily to treatment: deformities from hæmorrhages may be permanent.

Prophylaxis.—Fresh vegetables and fruit, fresh meat, and all substances containing the vitamin (*see* p 340) are preventive

Treatment.—

DIETETIC, viz, administration of the vitamin In severe stages, juice of fresh oranges or lemons most effective at least 3 daily Fresh meat juice less efficient. Preserved 'lime juice' is not anti-scorbutic No artificial mixture of salts is of any value, and all drugs are useless (*See* VITAMINS, p 340)

FOR THE MOUTH—Hydrogen peroxide washes Paint gums with silver nitrate solution (2 to 3 per cent) if condition severe.

CHAPTER LXVIII.

INFANTILE SCURVY.

A 'disease of deficiency' occurring in infancy, due to the absence from the diet of certain substances (vitamins), and characterized mainly by subperiosteal hæmorrhages and anæmia

Etiology.—Condition is identical with, or closely allied to, adult scurvy. Former names 'acute rickets', 'infantile rickets', and 'scurvy rickets' are erroneous, rickets resulting from absence of different vitamin: but rickets may co-exist, and both are 'diseases of deficiency'.

Anti-scorbutic Vitamin.—*See* Chapter LXIV, especially sections on 'Anti-scorbutic Vitamin' and 'Diets of Infants'.

Morbid Anatomy.—*Effusions between periosteum and bone.* May be fractures or separation of epiphyses. Structure of bone shows *rarefaction*: often hæmorrhages into marrow. Occasionally swollen gums, hæmorrhages in various tissues; rarely nephritis. No other constant changes. Rickets common.

Symptoms.—

PERIOD OF DEVELOPMENT.—Usually about age of 8 months. This accords with the period of development of adult scurvy. Infant on diet of proprietary foods or condensed milk.

ONSET of severe symptoms often sudden, but previous ill-health. Pale, but not wasted.

CHARACTERISTICS —

1. *Screams when touched or moved.* Extreme tenderness. Lies very still. Both legs often everted.
2. *Swelling*, usually lower end of femur: indefinite nature. Near but not in joint, extends up shaft, very tender; skin not hot, due to subperiosteal hæmorrhage. Less often, upper end of tibia. Upper extremities rarely.
3. Swelling of gums round teeth less constant than in scurvy. Gums rarely ulcerate. *If no teeth, gums normal.*

Occasionally —

4. Hæmorrhage into orbit and proptosis.
5. Fractures close to epiphyses, or separation of epiphyses.
6. Hematuria.

Very rarely, hæmorrhage in other sites, viz., skin and deeper tissues.

Anæmia of secondary type. Constant, but not extreme.

Temperature may be normal. Rarely above 102°.

Diagnosis. —

CHARACTERISTICS. (1) Screaming when touched. (2) Indefinite swelling at lower end of femur. (3) Predisposing diet. (4) Rapid cure when treated.

DIAGNOSIS FROM —

ACUTE OSTEOMYELITIS.—High temperature and severe constitutional symptoms.

RHEUMATIC FEVER.—Very rare under two years and never under one year.

INFANTILE PARALYSIS.—No swelling.

ULCERATIVE STOMATITIS.—Scurvy only affects *gums*.

Occasionally from acute leukæmia, chloroma, sarcoma of skull, renal sarcoma.

Treatment.—Condition yields readily to *dietetic* treatment. All drugs valueless. Give: (1) No proprietary or preserved foods. (2) Unboiled milk; (3) Orange or grape juice, four teaspoonfuls daily, or give raw meat juice. (*See* DIETS OF INFANTS, p. 341).

LOCAL TREATMENT.—Wrap limbs in cotton wool. Cage to keep off weight of bed-clothes.

CHAPTER LXIX.

PELLAGRA.

A disease of uncertain origin occurring in temperate and subtropical regions, characterized by lesions of the skin, alimentary canal, and nervous system, tending to be chronic, with recurrences in spring.

History and Distribution.—

Italy and Roumania for long formed chief centres. Also common in Mediterranean, and Balkan States.

In United States, recognized about 1907; now known to be widespread, especially in Southern States. In British Isles, described first in 1912 (Box and others). Found in South Africa, Nyassaland, etc. Wide recognition may be due to new occurrences or better diagnosis.

Etiology.—

AGE.—All ages; mainly 20 to 50 years.

SEX.—In United States females predominate. Otherwise about equal.

CLIMATE.—Mainly in warm climates.

SEASON.—Especially in *spring*; often yearly recurrence.

Poor and rural populations mainly affected.

Pathogenesis.—Almost certainly is a "disease of deficiency." Two main theories.—

1. A DISEASE OF DEFICIENCY.—Mainly occurs in persons largely subsisting on maize. Is not connected with milling of maize and removal of an embryo as in beri-beri, since whole maize is not preventive, nor is it due to spoiled maize.

   GOLDBERGER, 1914, reproduced pellagra in man by a diet low in protein and rich in carbohydrates.

   Probably results from a diet low in proteins, especially deficient in tryptophan and lysin, amino acids which are essential for human nutrition. These are present only in small amounts in zein, the protein of maize.

   Italian Government have strict laws against spoiled maize. 'Zeists' claim that those laws diminish pellagra. Antizeists emphasize that pellagra exists: (i) In spite of these laws, (ii) Where no corn is eaten, also not always where it is (pellagra is almost confined to poorer classes). On vitamin theory these objections are less potent.

2. PARASITIC THEORY.—

   a. Salmon Sections spread to *Simulium reptans*, buffalo gnat, which breeds in streams. But pellagra occurs at distance from running water.

b. Thompson-McFadden Pellagra Commission inclined to the infectious origin (but not *Simulium*), and detected no relation to diet

Note Attendants on pellagrins never contract disease no evidence of infection from one person to another

Morbid Anatomy.—

EMACIATION — Marked Bones fragile

BRAIN — Meninges thickened often œdematous

SPINAL CORD Meninges thickened *Posterior columns degenerated*, and Clarke's column cells degenerated. Most marked in cervical and dorsal regions

PERIPHERAL NERVES Changes rare

ALIMENTARY CANAL —Atrophy of mucous membrane Ulceration common in colon and rectum.

Liver, spleen, and kidneys small and fibrotic

Symptoms.—Protean Onset and progress insidious Course variable *Spring recidescence* marked symptoms improving in cool weather, and recurring yearly as summer approaches Affect mainly (1) Alimentary canal, (2) Skin, (3) Nervous system (4) Cachexia, dermatitis, and dementia Appear usually in this order Development may occupy many years and any one of these systems be mainly affected Here is a cinema of secondary type

Prodromal symptoms and three stages have been described, but are ill defined *

EARLY OR PRODROMAL SYMPTOMS Burning in mouth or extremities vertigo, insomnia dyspepsia, weakness Subsequent stages are briefly —

FIRST STAGE — Stomatitis and salivation Diarrhœa and dyspepsia Wasting Muscular weakness *Erythema* commences Insomnia, vertigo and irritability

SECOND STAGE *Erythema* recurs repeatedly also stomatitis and diarrhœa Mental symptoms develop melancholia, mania, tremors, etc

THIRD STAGE Cachexia advanced Dermatitis and diarrhœa. Death from cardiac weakness, exhaustion, or intercurrent disease.

Alimentary Canal.—

STOMATITIS Mucous membrane red and painful on improvement leaves a smooth tongue Occasionally ulceration of membrane *Salivation* profuse

DIARRHŒA — Severe and persistent Pain variable

PROGRESSIVE EMACIATION

Various gastric symptoms, anorexia, nausea, dyspepsia In later stages, atrophic gastritis with absence of free HCl

Skin Lesions.—

SEASON —Occur and recur in spring, abate after summer.

DISTRIBUTION —(1) *Backs of hands* earliest, and rarely escape,

* Osler and McCrae, *System of Medicine*.

Pellagra—Skin Lesions, *continued.*

spreads up forearm for varying distance; palm escapes. Other sites less commonly or later affected *face*, diffuse or butterfly-wing distribution; *neck*; feet; elbows; genitals. Aggravated by sun, but not always confined to exposed areas. (2) *Symmetrical*, always. (3) Sharp line of demarcation common

ERUPTION.—Commences as *erythema*, closely resembling sunburn Later, skin becomes swollen, dry, and desquamates, or wet pemphigoid lesions form After repeated attacks, skin is *pigmented*, dry, and thickened.

Summary—Symmetrical pigmented dermatitis of backs of hands, forearms, and face, with yearly recurrences.

Nervous System.—Changes usually slower and later than skin or alimentary system

MENTAL CHANGES—Irritability, melancholia, acute mania suicide not uncommon Progress to dementia

OTHER CHANGES—*Sensory system* burning pains, formication, girdle pains *Motor system* muscular weakness, ataxia tremors occasionally convulsions *Vertigo* common Knee jerks variable

Typhoid Pellagra.—An acute typhoidal condition may occur usually after several yearly relapses Mortality high, within one to three weeks

Pellagra sine Pellagra. Dermatitis absent Rare, but apparently authentic

Prognosis. In earlier stages, good with treatment Mortality in Italy about 5 to 10 per cent Prognosis depends mainly on mental changes with dementia, improvement slight *Duration*, often years death from exhaustion or intercurrent disease In typhoidal form, mortality high

Diagnosis.—Simple in fully developed condition in pellagrous countries Sporadic cases mainly found in asylums *Skin lesions* differentiate from sprue and scurvy

Treatment.—No specific known (1) *Change of diet give mixed full nutritious diet* (Pellagra almost confined to poor persons) (2) Change of climate to cool regions (3) Salt Italian government allows 17½ pounds yearly to each adult Symptomatic treatment for stomatitis, diarrhœa, etc., but results unsatisfactory. Arsenic and tonics. Protect affected areas from the sun.

Section V.—DISEASES OF THE DIGESTIVE SYSTEM.

CHAPTER LXX.

DISEASES OF THE MOUTH.

I. STOMATITIS.*

Clinical Groups.—(1) Acute (2) Aphthous (3) Ulcerative. (4) Parasitic: thrush (5) Gangrenous: cancrum oris, noma (6) Mercurial ptyalism (7) Other forms (8) Vincent's angina.

Acute Stomatitis (*Simple Stomatitis*)—

OCCURRENCE.—Frequent at all ages. Usually both general and local predisposing causes together. (1) General: debility, gastro-intestinal disturbance, specific fevers. (2) Local: in children, dentition; in adults, tobacco, carious teeth, and spiced foods.

SIGNS.—Mucous membrane in mouth dry and red. Tongue becomes swollen, furred, and indented.

SYMPTOMS.—Discomfort, especially on mastication.

TREATMENT.—

GENERAL.—Treat general condition, especially bowels.

LOCAL.—Sponge after each feed or wash mouth with hydrogen peroxide, or tincture of myrrh and borax. If obstinate, apply dilute silver nitrate (gr. ij to ounce).

INTERNALLY.—Pot. chlorate gr. ij to v, t.d.s., for child.

Aphthous Stomatitis (*Follicular or Vesicular Stomatitis*)—

OCCURRENCE.—In children, under 3 years, poorly nourished, especially after fevers, or gastric trouble.

SIGNS.—Multiple small ulcers with gray bases over inner surface of lips, cheeks, and edges of tongue; in severer cases also on pharynx. Commence as vesicles; ulceration rapid, usually within 24 hours.

SYMPTOMS.—Mastication painful; increased salivation; breath heavy; some constitutional disturbance.

TREATMENT.—*See* ACUTE STOMATITIS.

Touch, if possible, each ulcer with silver nitrate stick.

No special parasite associated. Heals rapidly.

Ulcerative Stomatitis.—

OCCURRENCE.—Children, after first dentition. Also epidemically in institutions. Predisposed to by malnutrition, and locally by irritation.

* See also DISEASES OF THE SALIVARY GLANDS, p. 162.

Ulcerative Stomatitis, *continued.*

SIGNS.—Commences at margin of gums: swelling, redness, bleeding, and then ulceration. Lips, cheeks, tongue swollen, but rarely ulcerated. Teeth may loosen, and rarely jaw necroses. Submaxillary glands enlarged. Salivation increased. Breath foul.

SYMPTOMS.—Mastication painful. Severe constitutional symptoms.

TREATMENT.—Potassium chlorate: very efficient. As mouth-wash (gr. x to ʒj): also internally gr. ij to v, t.d.s., child, or gr. v to x, t.d.s., adult. If severe, give anæsthetic and clean gums gently. General treatment.

No specific bacteria known.

Parasitic Stomatitis: Thrush.—

OCCURRENCE.—Mainly in bottle-fed infants, but also in debilitated adults. Predisposed to by local uncleanliness.

PARASITE.—A fungus, *Oïdium albicans*, or more correctly, *Saccharomyces albicans*—a yeast, with branching filaments and ovoid torulæ.

SIGNS.—Commences on tongue as dead white spots; entire buccal cavity may be covered by dry grayish membrane; grows among superficial epithelial cells, and scrapes off readily.

DIAGNOSIS from apthous stomatitis, by absence of ulcers, dryness, presence of membrane, and microscopic examination.

TREATMENT.—Often very resistant.

GENERAL.—Of great importance. Dose of castor oil. Clean bottles or teats.

LOCAL.—Apply gently glycerin and borax, or sodium sulphate (3j to ℥ij water), or sulphurous acid (diluted six times).

Gangrenous Stomatitis: Cancrum Oris: Noma.—Rapidly progressing infective gangrene. Rare. Is presumably bacterial, possibly sparochætal or anaerobic.

OCCURRENCE.—In children following acute fevers, especially measles; generally very debilitated and neglected subjects.

SIGNS AND SYMPTOMS.—Commences as sloughing ulcer usually on inner side of cheek; induration and gangrene proceed rapidly; cheek usually perforates; may involve bone. Constitutional symptoms very severe. Later, apyrexia and extreme toxæmia, accounting for absence of pain. *Death* almost invariable, 4 to 7 days.

TREATMENT.—Only effective treatment is widespread resection of tissues, and strong antiseptic. Insidious onset generally results in most cases being inoperable on first observation.

Mercurial Stomatitis; Ptyalism.—In susceptible persons, may follow very small doses.

SYMPTOMS.—Earliest, usually tenderness on biting. Salivation excessive; gums spongy, tongue swollen, breath foul, salivary glands enlarged; teeth may feel loose. In severe cases teeth drop out, and jaw necroses; rarely seen.

TREATMENT.—Stop mercury, open bowels, give alkaline drinks, liquid diet. Alkaline mouth wash.

Internally, potassium iodide said to be effective.
Recovery usually rapid.

Other Forms of Stomatitis.—

SORE MOUTH IN NURSING WOMEN.—Common. Small ulcers on lips and cheek.

Give tonics and good diet: glycerin and borax locally. Heals readily.

BEDNAR'S APHTHÆ.—Ulcers on hard palate of infants. Due to injury by artificial nipple or finger.

PIGMENTATION.—Occurs in Addison's disease.

MANY SKIN DISEASES, herpes, pemphigus, etc., may attack the buccal mucous membrane.

INFLUENCE OF STOMATITIS ON DEVELOPING TEETH.—'Erosion', pitting of teeth owing to defective formation of enamel, results from stomatitis in children, especially mercurial. Best seen in incisors. No relation to congenital syphilis.

ACUTE LEUKÆMIA.—The formation of a semi-acute localized sloughing area on the mucous membrane of the cheek is not uncommon and may be the earliest symptom. The blood should always be examined in such a condition, even if no spleen or glands are palpable. No surgical operation has any good effect.

Vincent's Angina.—An inflammatory condition of the fauces and mucous membrane of the mouth. Occurs at all ages, especially with pyorrhœa, syphilis, and other affections of the mouth. Is infectious by direct contact.

PATHOLOGY.—Two organisms present: (1) Vincent's spirillum: thin, two or more loose spirals: resembles *S. refringens*. (2) Fusiform bacilli: long tapering bacilli: Gram-negative: preferential anaerobe: possibly a stage of the spirillum (unproved). Streptococci and other bacteria usually present.

SYMPTOMS.—Vincent described two types: (1) Membranous or diphtheroid: adherent membrane, usually on tonsils but also on neighbouring structures: bacilli only present. (2) Ulcerative: small ulcers with broken-down membrane: bacilli and villa present.

Distinction of types generally indefinite. Inflammation usually involves, in addition to fauces, gums and mucous membrane of mouth; gums swollen and red, often small ulcers near line of teeth, bleed readily.

Onset usually sudden. Mouth sore, general malaise, headache, prostration, and irritability: pyrexia, usually moderate, may be absent. Cervical and submaxillary glands enlarged and tender: suppuration rare.

DURATION.—From a few days to two weeks. Occasionally becomes chronic.

COMPLICATIONS.—Many described: not common, but may be severe, e.g., otitis media, necrosis of tonsils or jaw.

DIAGNOSIS.—Difficult, it disease limited to fauces. Often depends on examination of smears. Wassermann reaction is always negative, unless there is coexistent syphilis (McKinstry).

Vincent's Angina, *continued.*

TREATMENT.—*Swab carefully, and wash mouth frequently with hydrogen peroxide solution.* Local cleanliness essential clean, but do not remove, teeth. Numerous drugs, local and internal, recommended, but no evidence of specificity Among others: potassium iodide internally, swab affected areas locally with salvarsan powder, sodium perborate gargle Keep bowels open Tonics later.

II. DISEASES OF THE TONGUE.

Apart from conditions noted under stomatitis, the following are important:—

Geographical Tongue (*Eczema, Erythema, Pityriasis of the Tongue: Wandering Rash*)—

Desquamation of superficial epithelium, starting from a spot and spreading in a ring central parts heal while periphery widens Fusion of various rings results in 'geographical' outline Fungiform papillæ may remain prominent May wander round tongue for months

ETIOLOGY.—Unknown No bacterium found No relation to syphilis important to avoid error of diagnosis

OCCURRENCE.—In infants, children, and also in adults usually with gastric troubles: often transient, but tends to relapse

SYMPTOMS.—Often none May itch, and in adults cause fear of cancer.

TREATMENT.—None of effect Mild mouth washes

Leucoplakia Buccalis (*Leukokeratosis Tylosis Linguæ*)—White patches on the tongue due to thickening of superficial layers of epithelium. Patches either smooth or fissured

ETIOLOGY.—Nearly, if not always, syphilitic with local factor e.g. carious teeth, excessive smoking

OCCURRENCE.—(1) Small raised white spots (2) Diffuse, patchy, bluish-white thickening of epithelium common type (3) Diffuse throughout buccal cavity rare.

May become epitheliomatous at any stage, shown by ulceration, induration, or nodules. May disappear spontaneously

TREATMENT.—Very obstinate Anti-syphilitic remedies useless Avoid all irritants. Try X rays Active local treatment is inadvisable

Black Tongue (*Hairy Tongue: Melanoglossia*) Black patch on centre of dorsum due to prolongation of filiform papillæ nature and origin of pigment unknown, not invariably black. May simulate hairs. No bacterium found. Treatment useless Returns if scraped off. Disappears spontaneously

Ulceration of the Tongue.—Causes: (1) Trauma, e.g., sharp tooth. (2) Stomatitis; ulcerative, aphthous, nursing women. (3) Syphilis. (4) Neoplasm. (5) Tuberculous. (6) 'Dyspeptic', if all other forms excluded.

III. FŒTOR ORIS.

(*Foul Breath.*)

Causes.—Numerous :—

MOUTH.—(1) Pyorrhœa alveolaris; (2) Decayed teeth; (3) Chronic lacunar tonsillitis (test by rubbing tonsil with finger), (4) Any form of stomatitis.

RESPIRATORY TRACT.—Numerous, e.g. :—

Nose.—Atrophic rhinitis, ozæna, suppuration in sinuses.

Lungs.—Bronchiectasis.

INDIGESTION.—Breath 'heavy', mainly due to condition of mouth.

EXCRETION of many substances by the lungs, e.g., acetone. Tobacco, onions, garlic, and certain drugs may affect breath.

Local conditions in the mouth account for most cases.

IV. THE TONGUE IN DISEASE.

The tongue in health is firm, red, moist, with slight fur posteriorly, in front of the circumvallate papillæ. Changes in ill health depend in the main on diminution of buccal secretions, occurring chiefly in: (1) Gastro-intestinal disturbance; (2) Pyrexia; (3) Local conditions, e.g., mouth breathing.

Principal changes are (a) Presence of fur; (b) Dryness.

PRESENCE OF FUR.—Fur is formed by matter accumulating on the filiform papillæ, mainly bacteria; also, in disease, desquamated epithelium and food. Forms normally during sleep especially posteriorly, being untouched by roof of mouth in rest. Especially forms after milk.

Fur does not form in infants owing to absence of filiform papillæ.

Occurs in any pyrexia, especially typhoid, rheumatic fever; in gastro-intestinal disturbance, such as morning tongue after heavy meal overnight. Due to diminution of saliva.

DRYNESS OF TONGUE. In severe toxæmia, especially ... to diminished saliva, and to mouth breathing from muscular weakness, seen in acute septicæmia, acute peritonitis, etc. If tongue has been furred previously, it becomes dry, brown, and shaggy. In very acute cases where no furring has previously occurred, tongue is dry, brown, and glazed. Moistening of the tongue is a good sign in prognosis. A similar tongue occurs also in cholera, and in severe diabetes but usual diabetic tongue is large and red ('raw beef tongue').

Enlarged papillæ may be prominent in children: especially in scarlet fever.

The tongue varies in different individuals. A chronic furred tongue appears compatible with good health. A clean tongue is certainly not proof of normal gastro-intestinal functions: is especially clean with hypochlorhydria.

CHAPTER LXXI.

DISEASES OF THE SALIVARY GLANDS.

I. PTYALISM OR SALIVATION: HYPERSECRETION.

'Salivation' is applied to an amount of saliva which causes escape from the mouth, or needs special effort to swallow: total amount not necessarily increased. Normal daily quantity 2 to 3 pints.

Causes.—

1. Gastric disturbance: especially before vomiting.
2. Local causes: (*a*) Amount increased: dentition, stomatitis, tic douloureux; (*b*) Difficulty in swallowing, e.g., laryngitis.
3. Mental and nervous affections, e.g., in melancholia and bulbar paralysis from impairment of swallowing. In rabies marked and viscid. May be functional.
4. During gestation. Occasionally with menstruation.
5. Mercurial ptyalism. Occasionally from other drugs: gold, pilocarpine, jaborandi, etc.
6. A functional form of excessive salivation.

Treatment.—Atropine: bromides: treatment of cause.

II. XEROSTOMIA OR DRY MOUTH: HYPOSECRETION.

Suppression of secretion of salivary glands. Mainly in women, old and nervous. Ascribed to premature senile atrophy of glands. Lachrymal may also be affected.

Treatment.—Remove carious teeth or any irritant. Locally, glycerin and borax. Pilocarpine may be tried.

III. INFLAMMATION OF THE SALIVARY GLANDS.

Specific Parotitis.—*See* MUMPS, p. 229.

Infective or Septic Parotitis, or Parotid Bubo. Occurs with dryness of mouth and absence of mastication: nearly always suppurates: infection most probably by the duct. Caused by:

1. Infectious fevers, especially typhoid.
2. Rectal feeding, most commonly for gastric ulcer.

Less commonly:—

3. Injuries and operations on the pelvic, genito-urinary tract.
4. Facial paralysis; diabetes; chronic metallic poisoning.

TREATMENT.—Prophylaxis important: cleanliness of mouth in associated conditions. Locally: leeches or hot fomentations, free and early incision for suppuration.

Chronic Parotitis.—Enlargement of parotids may occur in lead, potassium iodide, or mercurial poisoning: also in secondary syphilis and chronic nephritis. Rare sequel of mumps.

Mikulicz's Disease.—Painless chronic bilateral enlargement of salivary and lachrymal glands. Cases described probably include various conditions: blood changes, enlarged lymphatic glands and spleen are recorded. Mikulicz's original case died of acute peritonitis, the glands diminishing in size: microscopically showed small round cells, said to be lymphosarcomatous. Other cases chronic, with changes mainly of infiltration of the interstitial fibrous tissue. X rays should be tried.

In mumps, lachrymal glands may be enlarged: and in leukæmia parotid, submaxillary, and lachrymal glands simultaneously: such are not cases of Mikulicz's disease.

Gaseous Tumours of Stenson's Duct and parotid swellings occur in musicians and glass blowers.

CHAPTER LXXII

DISEASES OF THE PHARYNX.

I. ACUTE PHARYNGITIS.

Causes.—(1) Cold, especially after hot rooms. (2) Constitutional conditions, e.g., gout, rheumatism, dyspepsia. (3) Occurs in epidemic forms. Occasionally secondary syphilis. Trauma (in children). Is a cause of "sore throat": tonsil and soft palate often affected. Frequently part of a coryza.

Symptoms.—

LOCAL. Tickling in throat and irritation causing cough; pain on swallowing; secretion early diminished, later much thick mucus. Neck muscles often stiff; occasionally palpable glands. Inflammation may extend to larynx (hoarseness) and Eustachian tube (deafness).

CONSTITUTIONAL. Rarely severe. Slight fever and malaise.

PHARYNX.—Mucous membrane injected, swollen, and later covered with mucus.

Treatment.—Treatment as coryza often sufficient. Give aperient.

LOCAL.—Compress to neck, hot or cold. Inhalations (tinct. benzoin co. ʒj in pint of hot water), or spray with Dowell's solution. Paint pharynx with Mandl's paint, twice daily.

II. CHRONIC PHARYNGITIS.

Causes.—Numerous: (1) Tobacco or alcohol in excess. (2) Excessive use of voice, especially with faulty production or during acute pharyngitis; very common in clergymen. (3) Repeated

Chronic Pharyngitis—Causes, *continued.*

acute attacks. (4) Various constitutional conditions: gout, dyspepsia, chlorosis, climacteric.

Symptoms.—(1) Impairment of voice. (2) Frequent irritable cough, throat sore (3) Tenacious mucus in nasopharynx, much hawking; may be blood-tinged.

Varieties.—Three groups:—

GENERAL PHARYNGITIS.—Congestion and mucus on pharynx, soft palate, and uvula.

GRANULAR PHARYNGITIS.—Small round swellings of lymphoid tissue around mucous follicles on posterior wall, especially laterally; veins near distended; mucus present mainly in voice users.

PHARYNGITIS SICCA.—Mucous membrane dry and glistening

Treatment.—

GENERAL.—The general health and cause of condition need treatment, rest to voice; abstention from alcohol, tobacco and hot or spicy food; regulate digestion and bowels, change of air

LOCAL.—Sprays, e.g., water with common salt (ʒj in ℥x), tinged with permanganate Menthol pastilles

GRANULAR PHARYNGITIS Touch nodules with galvano cautery Subsequently Mandl's paint, twice daily

III. RETROPHARYNGEAL ABSCESS.

Suppuration in tissues between spine and posterior wall of pharynx

Occurrence.—

(1) *Healthy children*, age 6 months to 2 or 4 years Also not uncommon after measles and specific fevers

(2) Older children or adults *caries of cervical vertebra*

Symptoms.—(1) Malaise and pyrexia, altered voice, dyspnœa and dysphagia, (2) Head held back, chin forward, and mouth open

(3) Tumour in mid-line of pharynx, visible or easily palpable Glands on one side of neck usually enlarged

Prognosis.—Fatal not uncommonly, from œdema of larynx suffocation by inspiration of pus, or cellulitis of neck

Treatment.—Steam tent, or steam inhalations

EVACUATE PUS.—Methods: (1) Through mouth with guarded bistoury. *Head must hang back* over table to prevent pus entering trachea (2) Laterally behind sternomastoid muscle preferable Strict asepsis necessary Tracheotomy instruments to be kept ready, for œdema of larynx

IV. LUDWIG'S ANGINA.

(*Cellulitis of the Neck*)

Streptococcal inflammation of the deeper tissues of the neck Probably spreads from tonsils or other structures in mouth through deep cervical glands. Usually stout healthy adult males

Symptoms.—(1) Constitutional symptoms very severe. Mortality high. (2) Swelling of neck, brawny. Usually commences in submaxillary region. (Acute phlegmon of pharynx is a severer stage.)

Treatment.—Free and deep incisions in neck: actual pus rare, but may discharge later. (Œdema of larynx common, tracheotomy must be performed if dyspnœa. Vaccine should be prepared and employed.)

V. HÆMORRHAGE.

Occurs from: (1) Pharyngitis, (2) Epistaxis, (3) General hæmorrhagic conditions. Rarely profuse. Diagnosis from hæmoptysis and hæmatemesis.

VI. ŒDEMA OF UVULA.

Usually from chronic interstitial nephritis. May be very great. Rarely in debility.

CHAPTER LXXIII.

DISEASES OF THE TONSILS.

Classification.—(1) Acute tonsillitis. (2) Peritonsillar abscess (quinsy). (3) Chronic tonsillitis (chronic enlargement of tonsils). (4) Adenoids (enlargement of pharyngeal tonsil—included owing to close association).

I. ACUTE (FOLLICULAR) TONSILLITIS.

An acute bacterial infection of the lymphoid tonsils.

Etiology. All ages, commonest in childhood, rare in infancy. Three groups of cases: (1) *Sporadic.* Often following chill or exposure, especially in ill health or with chronic enlarged tonsils. (2) *Epidemic.* Not uncommon in schools, etc. Sporadic cases also are often infectious. (3) *Symptomatic.* Diphtheria, scarlet fever, measles, rheumatic fever and its allied conditions. Secondary syphilis closely similar. Diphtheria is only exceptionally follicular, and is dealt with separately.

Bacteriology.—Streptococcus is most common in severe types. Numerous organisms may occur.

Symptoms.—

LOCAL.—Sore throat; extreme dysphagia; pain often shoots to ears.

CONSTITUTIONAL.—Often severe, onset may precede sore throat. Shivering or rigor; pains in back and limbs; general malaise. Temperature 101° to 103°. Pulse rapid. Tongue furred, breath heavy. Constipation. Febrile urine.

Acute (Follicular) Tonsillitis—Symptoms, *continued.*

ON EXAMINATION.—Tonsils swollen and red: exudation in the crypts forming cheesy masses, confined to tonsils and easily removed without a bleeding surface. Rarely a diffuse membrane. Cervical glands often tender and palpable.

Duration.—Two to seven days.

Sequelæ and Fatalities.—Very rare in ordinary forms. Ludwig's angina may develop in severe streptococcal infections. In epidemics, occasionally definite sequelæ, e.g., endocarditis, pneumonia, paralyses.

Diagnosis.—In *diphtheria*, membrane is continuous, not necessarily confined to tonsils, leaves bleeding surface on removal; temperature not so high; dysphagia and sore throat less marked. But may exactly imitate, or be imitated by, follicular tonsillitis.

From *scarlet fever*, measles, rheumatic fever, no diagnosis can be made by examination of fauces.

Treatment.—Isolate patient, and before treatment swab throat for bacteriological examination.

GENERAL.—Saline aperients or mercury to obtain free motions.

LOCAL.—Hot fomentations to neck. Formamint lozenges to suck. Wash mouth frequently; and gargle, if possible, with hydrogen peroxide (10 to 20 vols.).

Sodium salicylate (gr. x to xx. t.d.s.) often used: also guaiacum. With pyrexia, tincture of aconite ♏ hourly until temperature falls to 100° (but watch pulse for weakening).

II. PERITONSILLAR ABSCESS.

(*Quinsy.*)

Suppuration in peritonsillar tissues; may include parenchyma of tonsil. Acute and chronic tonsillitis are predisposing causes, also general ill-health and exposure.

Symptoms.—

ONSET.—Local and constitutional symptoms as in acute tonsillitis, but more severe and prostration marked.

EXAMINATION.—Tonsils enlarged, red and œdematous, no exudation. One or both tonsils affected. May meet in mid line. Salivation marked. Cervical glands enlarged and tender. Spasm of muscles prevents opening mouth. *Fluctuation* develops in 2 to 5 days; palpable to finger; involves soft palate.

Treatment.—Hot fomentations to neck. Mouth-washes of hot water, or hydrogen peroxide (10 to 20 volumes).

WHEN FLUCTUATING.—Incise through mouth with guarded curved bistoury. Incisions in *soft palate* from above down and inwards towards uvula, to avoid vessels near tonsils. Subsequently, frequent mouth-washes.

Convalescence.—Not to be hurried. Tonics. Tonsillectomy for recurrent quinsy, but not in acute stages.

III. CHRONIC TONSILLITIS.

(Chronic Enlargement of Tonsils.)

Results.—(1) Interference with respiration, and tendency to mouth-breathing. (2) Liability to septic infections, whence acute tonsillitis, enlarged cervical glands, otitis media, peritonsillar abscess. (3) Liability to diphtheria; possibly also rheumatic and scarlet fever. (4) *Entrance for tubercle bacilli*; probable path to cervical glands.

Adenoids frequently but not invariably co-exist.

Treatment.—Tonsillectomy. General anæsthetic, administered carefully (lymphatism may be present).

IV. ADENOIDS.

(Hypertrophy of Pharyngeal Tonsil.)

Hypertrophy of the glandular lymphoid tissue normally present in the pharynx, sometimes called the "pharyngeal tonsil."

Even in a work of this character, emphasis must be laid on the enormous importance of this disease. As a blight on the health of a nation it stands, possibly even above tuberculosis. Nor is it irremovable. The importance of its diagnosis is only equalled by the necessity for its correct and complete treatment; too often casually and carelessly slurred over in the past.

Etiology. Age 5 to 10 year. Possibly more frequent in damp climates. Hereditary or familial factor common. Extremely frequent, and present in a high percentage of school children.

Morbid Anatomy. Red vascular irregular masses in the vault of the pharynx, formed of hypertrophied lymphoid glandular tissue, covered with epithelium. In older patients after recurrent inflammation fibrous tissue increasing growths firmer and bleed less readily. Size, appearance, and bulk increase.

Tonsils usually but not invariably enlarged. Chronic nasal catarrh usual.

Symptoms.—Result directly and indirectly from nasal obstruction and mouth breathing.

MOUTH BREATHING.—Often first symptom noticed by parents. Worse at night; snoring; noisy irregular respiration; disturbed sleep and nightmares. May be definite dyspnœa. Cough, dry and nocturnal, common without bronchitis. "Snuffing" common from nasal catarrh. Mucus often blood stained.

FACIAL APPEARANCE.—Characteristic "*adenoid facies*": Face lengthens, appearance expressionless. Nose pinched, alæ nasi fall in. Mouth open. Upper lip often retracted, hanging lower jaw. Roof of mouth raised and superior dental arch narrowed. Teeth irregular and often carious. Pallor common. Frequently deficient general growth.

HEARING.—Deficient. Very important. Due to (1) Extension of inflammation, (2) Obstruction of orifices by adenoids, and (3) Absorption of air in Eustachian tubes. Also (4) Otitis media common.

Adenoids.—Symptoms, *continued.*

VOICE.—Loses resonance; tone nasal. Substitution of letters *B* and *D* for *M* and *N*.

MENTAL CONDITION.—General dullness and inability to fix attention ('aprosexia'), resulting from above factors, e.g., deficient hearing, bad sleep.

RESPIRATORY TROUBLES.—Common: *bronchitis*, asthma, tracheitis, laryngitis. (Air inspired not warmed and moistened by nasal mucous membrane.)

SHAPE OF THORAX.—'*Pigeon breast*' common type: sternum prominent; Harrison's sulcus at attachment of diaphragm; section through thorax becomes triangular; costochondral junctions often depressed; occasionally lower end of sternum deeply depressed (funnel breast). Changes result from obstruction to inspiration and falling in of yet unhardened structures. With recurrent asthma, 'barrel chest' results.

CERVICAL LYMPHATIC GLANDS.—Often palpable from recurrent inflammation.

GASTRO-INTESTINAL DISTURBANCES and intestinal parasites are common. Probably associated with general overgrowth of lymphoid tissues.

VARIOUS SYMPTOMS.—Headache, fetor of breath. Nocturnal enuresis, asthma, habit spasms are often ascribed to adenoids.

Diagnosis.—By 'adenoid facies' and above symptoms. Adenoids not visible from mouth but palpable on digital examination. Tonsils usually enlarged.

Prognosis.—Good with efficient and early treatment. Atrophy usually occurs at puberty.

Treatment.

OPERATION.—Give general anæsthetic; remove tonsils first if necessary. Careful removal of adenoids. If completely performed recurrence rare. Symptoms rapidly improve.

General health, if deficient, should be improved *before* operation. *After removal*, tonics and attention to general health important. Chin strap at night, if mouth breathing persists. Systematic breathing exercises of great value.

CHAPTER LXXIV

DISEASES OF THE ŒSOPHAGUS.

I. ACUTE ŒSOPHAGITIS.

Except when origin is traumatic, is rarely of clinical importance.

Etiology.—

(1) Acute catarrhal œsophagitis: (*a*) In specific fevers, may extend from pharynx; (*b*) Idiopathic, in sucklings.

(2) Traumatic: foreign bodies, corrosives, etc.

Occasionally :—

3. Membranous : in diphtheria, and rarely in toxæmias.
4. Cancer and local disease.
5. Small-pox.

Morbid Anatomy.—In catarrhal form, mucosa swollen, covered with mucus. Phlegmonous inflammation may follow catarrhal form, or foreign bodies, or phlegmonous gastritis : gangrene may result.

Symptoms.—Pain on swallowing ; pain under sternum ; severity varies.

Treatment.—Ice by mouth, enemata ; morphia. Olive oil. For stricture after corrosives, dilate with bougies.

Œsophageal Varices.—Occur in chronic heart disease and cirrhotic liver. Rupture of vein causes hæmatemesis, or rarely melæna only : frequent in cirrhotic liver.

Catarrhal Ulceration.—Peptic ulcer may be present at autopsy in chronic vomiting : post-mortem formation not completely excluded. Ulcers also in cancer, aneurysm, corrosives, rarely in enteric, or in diphtheria and in debilitating diseases. May be dysphagia. Diagnosis by œsophagoscope only.

Rupture of Œsophagus.—Extremely rare accident. The rupture occurs transversely at lower end. Great pain. Always fatal. Never diagnosed.

II. SPASM OF THE ŒSOPHAGUS.

(*Œsophagismus*.)

Spasmodic Stricture.—Occurs idiopathically in hysteria, hypochondriasis, and similar conditions. It also occurs in rabies and pseudo-rabies. Two types exist :

(1) In Course of Œsophagus.—*In neurotic persons* : onset purely psychical, or attributed commonly to some foreign body swallowed ; globus hystericus common ; site of spasm not constant, position of arrest of bougie varying ; dilatation rare, as œsophagus rapidly rejects food which does not pass ; some wasting but not emaciation. Attacks may be transient at first, becoming more persistent. *Symptoms* : (1) Regurgitation of food and liquids ; (2) Spasm relaxes before bougie. *Treatment*. Passage of bougies. General. Warm food better than cold. *Prognosis*. Good.

(2) At Cardiac End of Œsophagus.—Is associated with so-called 'primary idiopathic dilatation of œsophagus'.

Idiopathic Dilatation of Œsophagus.—Generally affects lower two-thirds of œsophagus : spindle-shaped, narrowing to normal at diaphragm. Three important phenomena : (1) Muscular wall of œsophagus always hypertrophied. (2) Cardiac sphincter not hypertrophied, with rare exceptions. (3) Bolus of meal passes rapidly to cardia and, after a short pause, enters stomach.

Idiopathic Dilatation of the Œsophagus, *continued.*

MODE OF ORIGIN.—Disputed. Muscular hypertrophy proves presence of obstruction: negatives Mackenzie's theory of atony of the wall. Principal theories are:—

1. Failure of the co-ordinating mechanism which produces relaxation of the cardiac sphincter during swallowing (Hurst, "Achalasia of the Cardia"). *Physiology.* As each peristaltic wave passes down the œsophagus in the act of swallowing, a stimulus normally passes through a reflex arc to the longitudinal muscular fibres of the œsophagus, which then contract and open the cardiac sphincter, thus allowing the ingesta to enter the stomach. *Experimental production.* The longitudinal fibres are supplied by the vagus, and are paralyzed by its division (Langley, in cats), with subsequent feeding, if vagus be divided below the recurrent laryngeal nerve. Cannon has produced dilatation of œsophagus with muscular hypertrophy, viz., a condition similar to that occurring in man. *Conclusion.* Incoordination between the passage of a peristaltic wave and the contraction of the longitudinal muscular fibres which open the cardiac sphincter will explain nearly all instances of idiopathic dilatation.
2. Cardiospasm, viz., spasm of the sphincter fibres at lower end of circular coat of œsophagus. In this event hypertrophy of sphincter would be expected; certainly usually absent, but presence is recorded in a few instances.
3. Kinks of lower end of œsophagus. Presence in certain cases shown by œsophagoscopy and bismuth meals.

Other factors may be concerned, e.g., previous œsophagitis or ulceration.

DIAGNOSIS.—(1) Food said to "stick in the chest"; (2) Regurgitation of undigested food and liquid after some interval; (3) X-ray, with opaque meal.

TREATMENT. Gradual dilatation with bougies. Has been done through stomach successfully when due to kinks.

Paralysis of Œsophagus.—Occurs in certain central nervous diseases, e.g., bulbar paralysis, in hysteria, and in diphtheria. Very rare. Dysphagia without regurgitation.

TREATMENT.—Stomach tube, and treat cause.

III. ŒSOPHAGEAL OBSTRUCTION.

Causes.—

CONGENITAL ATRESIA.—(i) Œsophagus ends at bifurcation of trachea: lower segment arises from trachea or bronchus and opens into stomach. No case has survived. (ii) True stenosis very rare.

CICATRICIAL STRICTURE.—(i) Corrosives: site either high near pharynx, when food immediately regurgitated and no dilatation occurs, or low near diaphragm, when dilatation and

hypertrophy of œsophagus may follow (ii) Syphilis very rare diagnosis usually doubtful.

3 TUMOURS IN WALL
4 DIVERTICULA
5 FOREIGN BODIES
6 EXTRINSIC TUMOURS.—Aneurysm; lymphatic glands, neoplasms, etc
7 SPASMODIC STRICTURES.—*See* SPASM OF ŒSOPHAGUS In all organic forms spasm may also be present.

Diagnosis. (*a*) Food 'sticks in the throat' (when obstruction is low, probably from a protective spasm) (*b*) Regurgitation of unchanged alkaline food (*c*) Opaque meal and X rays are diagnostic (*d*) Bruit on auscultating deglutition is absent or altered (unreliable) Bougies are inadvisable

Most frequent causes in order are (1) Aneurysm, (2) Neoplasm, (3) Corrosives (history slow onset

Treatment. Varies with cause Following corrosives slow dilatation with bougies, at first twice weekly, later once a month

IV. CANCER OF THE ŒSOPHAGUS.

Occurrence. (1) Male 80 per cent 50 to 55 years (2) Growth primary (3) Epithelioma

Lumen is obstructed and dilatation and hypertrophy may occur above

Sites. (1) Level of second cervical vertebra, (2) Inlet of thorax, (3) Lower third Statistics vary, surprisingly, as to relative frequency but upper third least common

Symptoms. (1) Difficulty in swallowing progressive becomes extreme regurgitation of unaltered food (2) *Liquid* comes first (3) Pain variable may be constant occasionally absent Cervical glands may be enlarged

Complications. (1) Extension on into vital organs causes fistulæ to pleura, etc (2) Pressure symptoms e.g. on recurrent laryngeal nerve enlarged lymphatic glands may cause pressure.

Diagnosis.—From other causes of stricture By bismuth and X ray and by passage of bougie after excluding aneurysm Is the usual cause of dysphagia and emaciation in men over 50 years

Treatment. Early gastrostomy Duration 6 to 12 months Death usually from perforation

V. DIVERTICULA AND DILATATIONS.

Diverticula or Œsophageal Pouches are protrusions of part of wall of œsophagus Two types —

1. PRESSURE OR PULSION DIVERTICULA.—On posterior wall, at junction of pharynx and œsophagus here lumen is narrow, with cricoid cartilage in front, and posterior muscular wall weak Pouch formed is enlarged by food, becomes most direct continuation of pharynx, and pushes into neck, usually

Pulsion Diverticula of the Œsophagus, *continued*

on left from position of œsophagus. wall is thick, diameter of pouch several inches Usually in old age. Probably acquired and not congenital

Symptoms Progressive difficulty in swallowing Loud gurglings Pouch can be emptied by pressure *Treatment* Operation results good otherwise usually death from wasting

2 TRACTION DIVERTICULA On anterior wall, at bifurcation of trachea from cicatrization and contraction of adherent lymphatic glands Rarely exceed one inch No symptoms unless perforated by foreign body from food occasionally enlarged by pressure of food

Diagnosis —Bismuth meal and X rays

Dilatation of Œsophagus. -

1 PRIMARY IDIOPATHIC DILATATION —See p 499
2 SECONDARY TO STRICTURE

CHAPTER LXXV.

DISEASES OF THE STOMACH.

I. PHYSIOLOGY OF DIGESTION.

Mastication and Salivary Digestion. -

SALIVA is a slightly alkaline liquid containing, besides albumin salts and potassium sulphocyanate -

1 MUCUS Moistens food aids mastication and deglutition
2 PTYALINE (STARCH FERMENT) Hydrolyses starch to dextrin and maltose Rapidly destroyed by acid but with normal digestion and proper diet salivary digestion continues in the stomach for 30 to 40 minutes

Deglutition : Passage of Food from Mouth to Stomach.-

Three stages are described in deglutition, but division is artificial (1) To back of pharynx, (2) Through pharynx to œsophagus (3) Along œsophagus and into stomach During deglutition pressure in mouth rises to 20 cm of water and intragastric pressure falls to zero

When a semi solid bolus is swallowed, it passes freely to cardiac orifice without peristalsis, the walls of the œsophagus being lax apparently inhibited by act of deglutition With repeated acts of deglutition the inhibition persists the cardiac sphincter is also inhibited, and the food passes direct into stomach, at conclusion peristalsis occurs and empties tube of remnants With a single deglutition, delay may occur at cardiac orifice until peristalsis occurs.

TIME OF DEGLUTITION —For fluids and semi solids 4 to 10 seconds, average 6 For a well masticated solid bolus, 8 to 18

seconds. A dry bolus, e.g., capsule, usually hesitates at the arch of the aorta, and may occupy 15 or more minutes in reaching stomach.

AUSCULTATION OF FLUIDS.—Two sounds: (1) Impulse against posterior wall of fauces. (2) Gurgling into stomach 6 seconds later.

Gastric Juice.- Important constituents are —

1. PEPSIN - Hydrolyses proteins to proteoses, peptones, and lower molecules. Acts only in acid medium, preferably HCl.
2. HYDROCHLORIC ACID -- Functions: (a) Activates pepsinogen. (b) Controls pyloric sphincter (and partly cardiac sphincter). (c) Stimulates pancreatic secretion. (d) Hydrolyses starch and fat. Also antiseptic action.
3. MUCUS.
4. LIPASE - Hydrolyses fat to free fatty acid (aided by HCl).

Pepsin and HCl are only secreted by the tubular glands in the body of the stomach. These glands contain (a) Parietal cells secrete HCl. (b) Granular cells secrete ferments.

Secretion of Gastric Juice. Two factors concerned.

1. NERVOUS SECRETION (appetite juice). Commences within 5 minutes of food being taken into mouth. Of nervous origin through vagus, by stimulation of mucous membrane of mouth. Accounts for most of gastric juice.
2. CHEMICAL SECRETION (chemical juice). Commences 15 to 25 minutes after taking food. Mechanism: The food for early products of digestion stimulates pyloric mucous membrane which secretes a hormone gastric secretin; this is absorbed into blood stream and stimulates cells of glands of fundus to secrete, independent of all nerves.

INFLUENCE OF DIFFERENT FOODS, ETC., ON GASTRIC SECRETION -

1. Meat, meat extracts, impure peptones. Cause marked increase (especially HCl).
2. Milk, water. Slight increase.
3. Bread (unless previously digested with gastric juice), starch, white of egg, pure peptone. No effect.
4. Fats, sodium bicarbonate. Inhibit secretion.

Mustard, pepper, spices, alcohol cause marked secretion, also tea and coffee but variable in different individuals, also cigar after meals.

Shape of Stomach (in erect position).—The normal stomach is roughly tubular, and resembles a J in shape.

PYLORUS.- About one inch above umbilicus and slightly to right of mid line. Otherwise stomach is practically on left of mid line.

LOWER BORDER - At level of, or one inch below, umbilicus. Does not descend with respiration or on arrival of food.

UPPER MARGIN.- Occupies inner two thirds of left dome of diaphragm.

ŒSOPHAGUS.—Enters slightly below upper margin and towards the right.

THE AIR SPACE.- Area above entry of œsophagus contains air.

Physiology of Digestion—Shape of Stomach, *continued.*

In an empty stomach, this appears as a circular area; when stomach is full, lower level of air space is horizontal. Except for the air space, the walls of an empty stomach are in apposition.

Stomach consists of:

1. BODY OR FUNDUS. Roughly forms vertical arm of J; proximal two-thirds of stomach. Mucous membrane contains tubular glands secreting ferments and HCl.

   FUNCTION.—Especially secretory; peristalsis slight; absorption slight.

2. PYLORIC PORTION. Roughly horizontal; distal one-third of stomach. Divided frequently from the body by a strong contraction, the *incisura angularis*. Consists of (a) Pyloric vestibule or antrum; (b) Pyloric canal.

   FUNCTIONS.—(1) Mixing food and gastric juice (the "pyloric mill"); (2) Secretion of gastrin, secretin; (3) Absorption.

Movements of Stomach during Digestion. Observed after ingestion of opaque bismuth or barium meal.

When first portion of food enters stomach it streams rapidly down right side of fundus to pylorus without peristalsis; this portion maintains contents in a tubular form reaching to pyloric orifice.

As further food arrives until completion of an ordinary meal it is accommodated by increasing width to the left, separating the two walls in apposition. Thus the later food lies to the left of the earlier portions and is not immediately mixed with it.

Lower level of stomach should be clear in fasting meal.

PERISTALSIS commences very shortly after arrival of first food; waves commence slightly to left of incisura angularis, the fundus being little affected; as they proceed they increase in force and on reaching pylorus they make contractions. The constricted portion contracts and forces food against the pylorus and then back in a central stream to the fundus. The tone of the fundus regularly drives fresh portions towards the pylorus. The pyloric "mill" thus mixes the food and the gastric juice. The pyloric portion also absorbs certain products of digestion, of which the protein derivatives have the function of stimulating gastrin secretin, thus originating the chemical juice.

Note: The contractions are not preceded by a wave of relaxation as in ordinary peristalsis; they are myogenic in origin, and independent of nerves and of Auerbach's plexus.

OPENING OF THE PYLORUS. Acid on the gastric side of the pylorus has the function of relaxing the sphincter. Thus, when the concentration of HCl reaches a certain level, the pylorus opens and some chyme passes. Passage commences in about 10 minutes after arrival of food. Acid in the duodenum tends to close the pylorus.

THE STOMACH SHOULD BE EMPTY in 4 to 5 hours.

Effect of Gastric Digestion on Various Foodstuffs.—

FLUIDS.—No absorption by the stomach. Water taken alone, passes to the duodenum in one or two minutes.

PROTEINS.—Hydrolysed by gastric juice to proteoses, peptones, and to some extent to amino acids partly absorbed by the stomach protein digestion is never complete in the stomach Protein derivatives fix some HCl and thus prevent too rapid opening of pylorus by rising acid concentration

Vegetable protein demands more pepsin for digestion than animal protein

CARBOHYDRATES -Not digested in the stomach (but HCl can invert cane sugar) Carbohydrates, if taken at end of meal, pass to the farthest left of the fundus, where there is no peristalsis and no immediate mixing with other contents or with juice salivary digestion thus proceeds for 30 to 40 minutes Carbohydrates if taken at beginning of meal rapidly enter duodenum, since they do not fix HCl and the acid causes opening of pylorus. The normal arrangement of a meal—soup meat, carbohydrates—is thus physiologically correct

FATS. Partly hydrolysed to free fatty acids by lipase and HCl. Slowly [illegible] usually present in stomach 6 to 7 hours after ingestion This is attributed to (1) Inhibition of gastric secretion and slow rise of HCl, (2) Fatty acids on entering duodenum close the pylorus

Passage of Food through Duodenum : 'Duodenal Cap'.—Food commences to pass the pylorus in about 10 minutes, and forms the duodenal cap

The duodenal cap appears as a semicircular area placed like a cap over the upward turned pylorus and separated from it by a narrow band due to the translucent pyloric sphincter normally the outline is very regular

From the cap portions of chyme pass from time to time with great rapidity in a stream through the remainder of the duodenum to the jejunum.

Passage through Small Intestines.—The contents [illegible] rapidly through small intestine commencing to arrive in caecum in 4 to 5 hours after ingestion Owing to wide separation to mixture with juices and to rapidity of movements, the contents appear as a slight general opacity

Special research reveals two types of movements (1) 'Segmentation' or 'pendulum movements' Transient constrictions occur first at one point and then at another subdividing the gut and thus mixing the food and the juice, no propulsive effect (2) Peristaltic contractions Contents thus passed forward Finally a wave opens the ileocolic sphincter.

II. ACUTE GASTRITIS.

(*Acute Dyspepsia. Acute Gastric Catarrh*)

Acute inflammation of the mucous membrane of the stomach, resulting in gastric symptoms and varying constitutional disturbances, Often associated with entero colitis.

Etiology.—Occurs at all ages, but origin varies Common causes :—

(1) FOOD —'Errors of diet': (*a*) Quantity excessive : (*b*) Quality

Acute Gastritis Etiology, *continued.*

coarse, rich, or decomposing, food poisoning. Easily excited after previous starvation. Alcohol.

(2) SYMPTOMATIC.—Onset of acute infectious disease.

(3) TOXIC.—Irritant and corrosive poisons, viz., strong acids and alkalis, arsenic, phosphorus, etc.

(4) SPECIAL TYPES.—Phlegmonous and diphtheritic gastritis.

Predisposing causes include:—

IDIOSYNCRASY.—Common with individuals and families, either general or due to special articles of diet.

CHILDHOOD AND INFANCY. Especially from food, unripe fruit, and infectious disease.

Anæmia. Exposure to cold and wet, and extremes of temperature. Gout, chronic nephritis, diabetes, etc. Chronic gastritis and portal congestion from any cause.

Morbid Anatomy.—

MACROSCOPIC.—Mucous membrane swollen and hyperæmic with covering of mucus; may be hæmorrhages.

HISTOLOGY.—Swelling of mucous cells. Leucocytic infiltration.

GASTRIC JUICE.—Scanty. Increase of mucus. Usually diminution or absence of HCl; rarely increased.

Symptoms.—Vary greatly with cause and severity.

MILD TYPE.—Abdominal discomfort, anorexia; furred tongue; nausea; vomiting, giving relief. Headache common. Apyrexia, or rise slight. Duration 24 to 48 hours.

SEVERE TYPE.—Onset sudden; may be slight rigor. Fever rise to 103°. 'Nasty taste in mouth', conjunctiva dull, tongue furred, breath heavy, anorexia, thirst. Headache, giddiness, and mental inertia. Extremities cold. *Vomiting* of food, then bile. Acid *eructations*, may cause 'heart-burn' or set teeth on edge. Epigastric tenderness, distention of stomach by gas. Constipation, or not uncommonly diarrhœa. Urine of febrile type. Duration 1 to 3 days. Depression for several days.

Diagnosis.—Differential diagnosis of simple from symptomatic forms often impossible.

ACUTE INFECTIOUS DISEASES.—Suggested by pyrexia and *absence of dietetic error*, specially in children.

INTRACRANIAL DISEASE.

PERITONITIS, INTESTINAL OBSTRUCTION, APPENDICITIS.—Abdominal physical signs. Vomiting usually more marked.

TABETIC CRISES.—Test for evidence of syphilis.

GALL-STONE COLIC.—Distribution and character of pain.

TOXIC CAUSES, e.g., arsenic.—Special tests of gastric contents, etc., when poisoning suspected.

Numerous causes of gastric disturbances: catarrhal jaundice, gastric and duodenal ulcers, etc.

Treatment.—

MILD FORM.—Low diet. Castor oil or calomel.

SEVERE FORM.—*Indications*: (1) Remove irritant from stomach; (2) Rest for stomach.

GENERAL.—Warmth, especially to extremities. Mustard leaf or poultice to epigastrium, if tender. Hot water freely.

DRUGS.—Calomel, gr. ij to iij (adult), followed by saline purge. If diarrhœa, castor oil (ʒss) with tinct. opii ♏x.

VOMITING.—Aid by warm water, several tumblers; tickle fauces if necessary: repeat: eases acid eructations, nausea, and vomiting. Stomach wash if severe, and acid, hydrocyanic dil. ♏iij to v, with bismuth.

DIET.—Soda-water, as desired, or lime-water. Later, diluted milk.

PERSISTENT DIARRHŒA.—Mist. cretæ (B.P.), adding tinct. opii ♏v to x if necessary, or pulvis cretæ aromaticus c. opio gr. xx to xxx, t.d.s.

WITH IMPROVEMENT.—*Diet.* Farinaceous foods, tea, boiled fish. Avoid fats. *Drugs.* As gastric sedative:—

℞ Bismuth Carb. gr. xx; Mucil. Trag. q.s.
Bicarb. ʒ xx; Aq. Menth. Pip. ℥j
t.d.s. p.c.

TOXIC CASES.—Special treatment, depending on cause.

Prognosis.—Chronic or subacute gastritis may follow repeated attacks. Immediate recovery usually complete.

MEMBRANOUS OR DIPHTHERITIC GASTRITIS.

Very rare. Usually in children. No primary form. Secondary process, rarely in diphtheria, more often in small pox and other fatal debilitating conditions. No peculiar symptoms.

PHLEGMONOUS GASTRITIS.

Diffuse Phlegmonous Gastritis. Rare. Widespread inflammation of *submucosa*, usually by streptococci, i.e. cellulitis of stomach. Rarely any obvious cause; occasionally puerperal fever, more common in males.

MORBID ANATOMY.—Walls thickened, homogeneous red jelly appearance, very friable. Peritoneal adhesions and inflammation present. *Histology.* Œdema and marked cellular infiltrations, especially near pylorus; mucous membrane but slightly affected. No collections of pus.

SYMPTOMS.—*Severe sepsis with abdominal symptoms.* Onset sudden, often rigor. *Pain* in upper abdomen, rigidity and tenderness. *Vomiting* early. High temperature, rapid pulse, and marked constitutional disturbance. Tender *tumour* may be palpable. *Acute peritonitis* if life sufficiently prolonged. Stomach may rupture.

PROGRESS AND PROGNOSIS.—Collapse increases. Condition as in acute septicæmia. Fatal in few days.

Circumscribed Type.—*Localised abscess* of stomach wall. Very rare. Usually cancer present. Has been successfully evacuated.

III. CHRONIC GASTRITIS.

Chronic inflammation of the gastric mucous membrane, with resulting changes in the gastric juice and gastric functions.

Etiology.—Sequel to acute gastritis, usually recurrent attacks. Other factors as described under DYSPEPSIA.

Morbid Anatomy.—Varies greatly. The early stage is probably hyperæmia and congestion of mucous membrane with usual inflammatory changes. This may progress towards either (1) hypertrophy of mucous membrane especially near pylorus (hypertrophic gastritis), or (2) fibrosis of connective tissue and atrophy of mucous membrane (atrophic gastritis, finally achylia gastrica). Both forms and intermediate types are common.

Even when mucous membrane in fundus is thin, near pylorus it is often rough and irregular, and sometimes appears sufficient to produce some obstruction and partially account for dilatation (polypoid gastritis).

In common types (simple chronic catarrhal gastritis) the mucous membrane of fundus is usually thin, smooth, gray, and often pigmented; mucus excessive; veins may be distended; and ecchymoses and hæmorrhages present; at pylorus mucous membrane is rough and with many rugæ. Size of stomach normal or somewhat dilated.

The mucous membrane at pylorus is always thick in all types, with increase of HCl (sthenic gastritis).

Symptoms.—Epigastric discomfort or pain. Flatulence. Vomiting at intervals. Appetite diminished or occasionally craving. Acid eructations. Tongue furred. Bad taste in mouth. Complexion and conjunctivæ muddy. Intellect dull. Depression, irritability, giddiness. Constipation, occasionally diarrhœa.

Gastric Contents.—Included under three forms:

(1) *Simple gastritis.*—HCl diminished; ferments present, may be scanty.

(2) *Mucous gastritis.*—HCl diminished; ferments present, may be copious.

(3) *Atrophic gastritis (achylia gastrica).*—HCl and ferments completely absent.

Treatment.—See DYSPEPSIA.

IV. DYSPEPSIA.

Consciousness of the activities of the stomach. Physiological appetite forms an exception.

General Considerations.—Dyspepsia is not a morbid entity, but a group of symptoms due to various physiological or pathological activities of the stomach. Normally there is no consciousness of the presence or absence of food in the stomach, except a physiological degree of appetite and a feeling of pleasant repletion. Three groups may be recognized: (1) *Organic disease*: cancer, ulcer, dilatation, also visceroptosis, perigastric adhesions, and chronic gastritis. (2) *Functional affections*: no obvious anatomical changes. (3) *Neuroses of the stomach.*

Organic Disease.—Cancer, ulcer, and dilatation are considered separately. Chronic gastritis is ill defined from certain 'functional' types with hyper- or hypochlorhydria.

Neuroses. Are sometimes recognizable in presence of definite neurotic, hysterical, or neurasthenic evidence. Even in absence of such, certain forms of dyspepsia are commonly regarded as neuroses. (*See* NEUROSES OF THE STOMACH, p. 400.)

If hypochlorhydria be always regarded as chronic gastritis, and hyperchlorhydria as a neurosis, 'dyspepsia' becomes eliminated, but with present incomplete knowledge this is unjustifiable, and the term is retained here.

'Dyspepsia,' therefore, includes numerous cases classifiable either as chronic gastritis or functional forms on the one hand, or as functional forms or neuroses on the other hand. Temporary attacks of dyspepsia similarly merge into certain forms of 'acute gastritis,' *viz.*, from errors of diet, but 'dyspepsia' is never applied to severer forms, e.g., toxic poisoning.

In the remainder of this section organic forms (except gastritis) and neuroses are not referred to except where mentioned for completeness, as in etiology.

GENERAL ETIOLOGY.*

Causes are (1) Physical habits. (2) Dietetic. (3) Local disease of stomach and other organs. (4) Constitutional diseases. (5) Neuroses.

Physical Habits of Life. (1) *Imperfect mastication.* Especially deficient and carious teeth. Hurried meals. (2) Irregular meal times. (3) Deficient exercise. Work immediately after meals (physical or mental). Over-exertion. (4) Constipation. (5) Bad cooking. Dirty cooking utensils.

Dietetic.—(*Mainly excesses.*) (1) *Alcohol.* Spirits. Light wines, owing to acidity. New beer: fermentation incomplete and continues in stomach. Spirits act specially on liver; wines and beer mainly on stomach. (2) *Tea.* overdrawn. Tannin hardens meat fibre. Appetite, physiologically correct, dictates tea for farinaceous and not for meat meals. (3) Excessive fluid during meal. (a) Dilutes gastric juice. (b) Softens food with water instead of saliva and ferments, and aids insufficient mastication. (4) Excess of solids. (5) Fats in excess, possibly, as no digestion of fat occurs in stomach. When splitting of neutral fat occurs in stomach by action of bacteria, resulting butyric acid irritates. Free butyric acid is present in rancid butter. (6) Heavy pastry, hot bread, pies. Common in America. (7) Sugar in excess. Causes over-secretion of mucus. *Various other articles*: Tobacco in excess. Fruit, over-ripe or unripe. High meat. Chewing tobacco (excessive salivation). Vinegar in excess. Power of digestion varies greatly in different individuals. *Idiosyncrasies* to various articles are very common and often hereditary.

* *See* Allbutt and Rolleston's *System of Medicine*, Vol. III.

Dyspepsia - General Etiology, *continued.*

Local Disease.—

STOMACH.—Cancer; ulcer; dilatation; visceroptosis; adhesions.

OTHER ORGANS.—(1) *Liver: cirrhosis* impedes gastric circulation. (2) Chronic heart disease: through portal circulation, also 'epigastric angina'. (3) Gall bladder. Remoter organs: occasionally appendix (*see* p. 419), colitis, movable kidney, uterus.

Constitutional Diseases.—

PHTHISIS.

NEPHRITIS, GOUT, ANÆMIA, and all debilitating diseases.

Neuroses.—*See* NEUROSES OF THE STOMACH, p. 400.

Common Causes without Grave Illness. (1) Deficient teeth. (2) Alcohol. (3) Anæmia. (4) *Early phthisis.*

[NOTE.—The symptomatology and treatment below do not refer to organic disease, or to neuroses of the stomach.]

GENERAL SYMPTOMS.

Often chronic, but intermissions and variations in intensity occur.

Summary of Symptoms.

1. Epigastric discomfort. Varies from oppression to acute pain.
2. Flatulence. Also acid eructations; water brash.
3. Nausea and vomiting. Latter rarely prominent.
4. Alteration of appetite. Usually diminished.

Other Symptoms.—Sallow complexion; conjunctivæ muddy. Tongue furred. Bad taste in mouth. *Teeth* carious or deficient. Constipation. Occasionally diarrhœa. Cough usually from pharyngeal mucus. Temperature normal. Pulse often slow; may be palpitations. Giddiness. Often mental depression and inertia, irritability, frontal headache, cold extremities.

(1) **Epigastric Discomfort: Pain.**—Varies greatly in degree. Oppression and fullness mainly from flatulence and distension, viz., in flatulent dyspepsia. May be severe pain. Precordial or localized sternal pain from acidity ('heart burn'). No superficial tenderness. Pressure usually increases pain.

(2) **Flatulence.**—Gastric flatulence occurs in—

FLATULENT DYSPEPSIA.—(1) Usually in middle age, with deficient teeth and hypochlorhydria. (2) May be a prominent symptom in hyperchlorhydria, from swallowing air. (3) Less constant in dilatation of stomach.

ERUCTATIO NERVOSA.—Pure neurosis; swallowed air.

CARDIAC PAIN, ANGINA, GALL-STONE COLIC.—Origin doubtful: air probably swallowed. Cessation of pain often associated with copious eructation.

INTESTINAL FLATULENCE.—Mainly from fermentations. Often with gastric flatulence.

ERUCTATIONS.—Relieve gastric, but not intestinal flatulence.

ACID ERUCTATIONS —May be: (1) Organic acids in hypo hlorhydria and flatulent dyspepsia; (2) Hyper hlorhydria

HEARTBURN (OR CARDIALGIA)—Due to irritation of acids on œsophagus: may follow a bland fluid, e g, tea, from increasing distention of stomach and relaxation of cardia Eased by fluids, whence swallowing of saliva Contractions of the œsophagus may be a factor of the pain

WATER-BRASH —The irritation of acid eructations and heart burn may cause excessive swallowing of saliva, which is brought into mouth as clear fluid, slightly alkaline to litmus

PYROSIS —Properly a strongly acid fluid brought up to mouth from the stomach, much rarer.

ORIGIN OF GAS IN STOMACH —

SWALLOWED AIR —(1) With food considerable with deficient mastication More with fluids than solids. (2) With saliva amount considerable, hence flatulence in pharyngeal irritation (3) Neurosis whence 'nervous eructations'. Also in heart attacks

BACTERIAL FERMENTATION In hypochlorhydria, mainly from cellulose, as in cabbages Of important clinical importance dilatation, or delay of food from any cause, otherwise stay of food in stomach is insufficient for much gas formation

Small amounts from action of HCl on ingested carbonates Possibly also from intestines and from regurgitation of alkaline pancreatic juice Origin by escape from blood is doubtful

Nature of Gases CO_2 oxygen and hydrogen also nitrogen if air is swallowed With fermentation, CH_4 Rarely H_2S Occasionally inflammable

3. Vomiting.—

FREQUENCY —Varies, but rarely a prominent symptom in organic dyspepsia Results from (1) Condition of stomach contents excessive quantity, irritative, decomposition (2) Condition of walls, irritated and hypersensitive

TIME OF VOMITING —(1) Early morning especially if alcoholics, much mucus (2) After meals at varying intervals When *immediately* after ingestion, usually neurosis

CHARACTER OF VOMIT Food in various stages of digestion mucus common Acids, both character and amount, depend on type of dyspepsia

Swallowing saliva in excess may induce vomiting either early morning or after meals the saliva resulting from dyspepsia, or from catarrh of pharynx

NAUSEA common.

4. Appetite.

ORIGIN OF APPETITE —Depends on: (1) Condition of stomach wall, (2) Gustatory nerves of mouth, (3) Indirectly on the needs of the body Direct cause probably depends on circulation of blood through stomach, with reflexes from distention of lymph spaces.

VARIATION IN THE CIRCULATION —(1) Diminished, in chronic

Dyspepsia—General Symptoms - Appetite, *continued.*

forms of dyspepsia and gastritis : whence deficient appetite. (2) Increased, in acute forms or exacerbations.

GASTRIC IRRITATION.—Appetite depends also on degree of gastric irritation : (i) *Mild*, appetite increased (small doses of arsenic). (ii) *Marked*, appetite craving, but small amount of food causes nausea, may occur at onset of bilious attack. (iii) *Severe*, anorexia and nausea.

APPETITE IN DYSPEPSIA thus may be :—

(5) DEFICIENT.—From : (1) Diminished circulation : usual form. After commencing to eat, may improve from increasing circulation (indication for tonics, local and general, exercise, etc.). (2) Severe irritation : food causes nausea.

(6) CRAVING.—From irritation : but small amount of food causes nausea (indicates sedative treatment).

Note.—Appetite good, but satiated by small amount of food, occurs in dilated stomach, and also in hypertrophied stomach with pyloric obstruction.

SPECIAL TYPES.

Dyspepsia may be classified correctly as regards (1) Pathological changes in motility and secretion, ascertained by bismuth and test meals, (2) Clinical symptoms, e.g., flatulence, neurosis, (3) Etiology, e.g., dietetic. In practice, certain types are distinguished mainly on symptoms : (1) Acid, (2) Flatulent, (3) Atonic, with dilatation (*see* p. 390) ; (4) Nervous (*see* p. 400) ; (5) Achylia gastrica.

Acid Dyspepsia.—Dyspepsia especially associated with 'heart burn', acid eructations, and sometimes flatulence and 'water brash'.

Acids in excess may be (1) Organic acids, (2) Hydrochloric acid.

ORGANIC ACIDS.—Hypochlorhydria. *See* FLATULENT DYSPEPSIA.

HYPERCHLORHYDRIA.—Excess of HCl occurs in (1) Peptic ulcers. Not further referred to here. (2) In nervous individuals chlorotic, and with definite neurosis. In nervous persons HCl generally high, but gastric symptoms not necessarily present. Sometimes neurotic signs predominate over gastric, justifying diagnosis of 'gastric neurosis', as in rare gastro mucorrhœa. Bacterial fermentation is not excessive, hence flatulence not marked unless air swallowed, as with increased saliva.

SYMPTOMS.—Patient usually plump, and teeth good. Tongue clean. Recurrent attacks at intervals. Symptoms in definite relation to food (½ to ¾ hours after). (1) Appetite often good, but 'afraid to eat'. (2) Epigastric discomfort or acute pain ; (3) Acid eructations ; (4) Vomiting may be frequent. Constipation common. Flatulence variable. Pain relieved by : (*a*) Food, fluid, and alkalis ; act by diluting or neutralizing acid, or clearing œsophagus. (*b*) Emptying stomach by vomiting, or by passage of food through pylorus.

Flatulent Dyspepsia (*see also* FLATULENCE, p. 380).—Associated specially with *hypochlorhydria* and *increase of organic acids*.

ETIOLOGY.—With diminution of HCl, bacterial fermentation occurs, with production of (1) Organic acids: butyric, lactic, and acetic. (2) Gases: Butyric acid appears specially irritating.

TYPE OF PATIENT.—Middle age, with deficient teeth. Poor physical and general health. Pallid, constipated, and cold.

SYMPTOMS.—(1) Flatulence and epigastric discomfort. (2) Appetite poor. (3) Acid eructations and 'heart burn'. (4) 'Water-brash' less often pyrosis.

'*Flatulent dyspepsia*' applies specially to this type, but note that the symptoms of apparent 'acid dyspepsia' (acid eructations, heart burn) result here from the effects of hypochlorhydria, the frequent cause of such symptoms in patients of this type: this effect is a cause of confusion. Such a case may be diagnosed as 'flatulent dyspepsia' (from the symptoms), 'hypochlorhydria' or 'anacid dyspepsia' (from analysis of gastric contents), or as often occurs, 'acid dyspepsia' (from the occurrence of heart burn and (organic) acid eructations).

Achylia Gastrica.—Diagnosed only by gastric analysis: absence of gastric secretion: (a) no HCl, (b) no ferments. Occurs in: (1) Atrophy of mucous membrane: chronic dyspepsia, severe anæmia, pernicious anæmia. (2) Neurosis: achylia gastrica nervosa. Rare.

Symptoms with atrophy: Severe pain, vomiting, and wasting. If motility of stomach good, symptoms may be slight.

DIAGNOSIS from carcinoma: (1) Long duration. (2) Complete absence of HCl and ferments, and very low organic acidity.

DIAGNOSIS.

Often very difficult. X rays in doubtful cases. From:—

ORGANIC DISEASE OF STOMACH:—

CANCER: Short duration, rapid wasting, frequent vomiting, may be hæmatemesis. HCl absent from gastric contents. Tumour decisive.

ULCER: Hæmatemesis. HCl in excess in gastric contents. Superficial tenderness.

DILATATION: Inspection of abdomen.

DISEASE OF OTHER ORGANS: Gall bladder, appendix, liver (cirrhosis), heart.

CONSTITUTIONAL DISEASES: Especially phthisis.

TREATMENT.

Preliminary to treatment, investigate: (1) Cause: *see* GENERAL ETIOLOGY. (2) Type: based on symptoms and signs, and, when possible, analysis of gastric contents and X ray after bismuth meal.

General Principles.—(1) Remove cause, if practicable. (2) Diet suitable to condition. (3) Therapeutic and other measures.

Note.—(a) Teeth and constipation are primary considerations for treatment; also the encouragement of a good circulation. (b) Hyperacidity—inorganic or organic—present in great majority.

Dyspepsia —Treatment, *continued.*

and needs alkaline treatment, at least as preliminary. (c) Neurotic factor common (d) Avoid all drastic treatment.

REMOVE CAUSE.—In general —

1 Teeth to be attended to, and general condition of mouth

2 Mastication to be slow and perfect. Meals at regular hours Those who dine alone, and no others, should read at meals, to prevent undue rapidity But, as we are told, "a generous meal consumed with mirth, is better than a physician's prescription in the solitude of the chamber" (Allbutt)

3 Regular exercise. Rest before and after meals Avoid over-fatigue Walking home after day's work needs *10 minutes rest before dinner.*

4. Bowels open regularly Not occasional purges

5 Warmth to abdomen and feet Avoid chill after chief meals Smoking only after meals

DIET.—Chart of hours and diet essential, noting patient's preferences, and cause and type of dyspepsia

DIGESTIBLE ARTICLES —Chicken, mutton, game (comparative absence of fibrous tissue) Boiled fish, especially whiting and sole (short muscle fibres) Spinach, asparagus, cauliflower. Farinaceous foods (except as below) Toast

Minced meat lightly cooked is easily digestible (Vegetable protein needs more pepsin)

INDIGESTIBLE ARTICLES —Pork, beef Twice cooked or over cooked meat Condiments. Fried fish Cabbages (especially with flatulence) New bread Brown bread Short pastry, pies and tarts

FATS —Except fresh butter, in strict moderation no fat foods or fat soups

SUGAR —Restricted

FRUIT —Stewed fruit good for constipation Rhubarb, strawberries, and tomatoes contain excess of acid salts

FARINACEOUS AND PROTEIN FOODS —Roughly at separate meals, viz., farinaceous at breakfast and tea (times of digestion vary) Avoid tea at protein meals Chief meal midday or evening, as is most convenient for resting

FLUIDS —At least 2½ pints daily Hot water, ½ pint, sipped an hour before meals, including breakfast (and at night), amount during meals diminished (In atony and dilatation amount of fluid must be diminished especially during meals)

ALCOHOL —Allowed sparingly in hypoacidity, not with hypersecretion. Avoid all acid wines. Small amounts of alcohol aid digestion, as seen in 'sherry and bitters' before meals and liqueurs after. To be advised with caution.

MILK DIET.—Strict milk diet rarely indicated; in general not advisable and by no means always well borne. Short period with severe dyspepsia, especially with nephritis and portal obstruction (cirrhosis and heart disease). Diluted preferably. Peptonized if necessary. Definite rules to be laid down

(*a*) Times interval 3 hours (*b*) Quantity 3 pints daily (Contraindicated in dilatation.) *Watch stools* for undigested curds if present, reduce milk, and add eggs and toast.

PAPPY SEMI-SOLID STARCH FOODS. Rarely advisable in any type of dyspepsia, being swallowed without mastication and with consequent loss of salivary digestion.

THERAPEUTIC MEASURES. (1) Replace deficiences in gastric juice. (2) Neutralize excesses and give gastric sedatives. (3) Stimulate secretion. (4) Constitutional remedies.

REPLACE DEFICIENCIES. Specially in atrophy of mucous membrane and in common mild dyspepsia with suggestion of neurosis. (1) *Hydrochloric acid*: acid hydrochlor. or nitro-hydrochlor. dil. ♏xx p.c. or acidol gr. xv in water. (2) *Digestive ferments*:

Pepsin: In tablets gr. v to x, or combined with acid —

℞ Glycerin ℥j Glycerin ♏xv
Acid Hydrochlor. Dil. ♏xx Aq. ad ℥ss

Take 15 minutes p.c.

Pancreatin: In tablets with food, half hourly 2 hours p.c.

NEUTRALIZE EXCESSES.

Alkalis: neutralize hyperchlorhydria or HCl or organic acids also action. (1) Sedatives to stomach with bismuth. (2) Stimulants to secretion (food, bicarb. before meals).

Sedatives: in hyperchlorhydria and pain. Sodium bromide, bismuth, dilute morphine before meals.

STIMULATE SECRETION AND PERISTALSIS. MOTILITY. Indicated in hypochlorhydria in certain forms of dyspepsia with flatulence and constipation.

Pepsin: [illegible] rhubarb often added.

Uncharacterized: in small quantity.

CONSTITUTIONAL REMEDIES. Tonics, iron and quinine. Alginate of iron, gr. x, is useful in chronic constipation.

LAVAGE: Useful if much pain, vomiting, flatulence, or dilatation, once daily, morning or evening, on empty stomach. Sod. bicarb. (℥j to pint). Large amounts until washings are clear. (*See* DILATATION OF STOMACH.) Patient should not acquire habit himself if possible.

MASSAGE, ELECTRICITY, HYDROTHERAPY. With suitable and debilitated cases.

SPAS (e.g., Vichy). The routine is often valuable.

Treatment of Special Types and Symptoms.

Hyperchlorhydria. Attention to general health, tonics, rest, and drainage. Salines if constipation.

DIET.—Meat and protein; plenty of fat (inhibits gastric secretion); avoid carbohydrates. No soup or meat extracts. No alcohol. Salisbury diet useful: meat, half raw, minced (3 to 4 oz. t.d.s.), with stale bread and butter, hot water, butter and cream.

Dyspepsia—Treatment of Hyperchlorhydria, *continued.*

DRUGS.

(1) *Sedatives before meals* :—

℞ Sod. Brom. gr. v) Aq. Chloroformi ad ℥ss
Bismuth. Oxycarb gr. x

(2) *Alkalis after meals* (½ hour) —

℞ Sod. Bicarb
Bismuth. Oxycarb. } āā gr. xx (in water or milk).
Magnesii Carb.

Similar fluid prescriptions are efficacious, but have a large deposit, and addition of mucilage causes an unpleasantly thick draught.

Avoid *bitters and acids*

For flatulence : carminatives (*see* next section).

Hypochlorhydria, Flatulent Dyspepsia.—Special attention to teeth, constipation, and general principles of treatment

DIET - Give especially soup and meat extracts, minced meat (lightly cooked), fish, eggs toast rusks Meals otherwise dry, to ensure mastication fluids at end of meals Avoid fat, except butter in moderation Diminished farinaceous foods, potatoes, cabbage No pastry, sugar, or strong tea Little whisky or brandy allowable

DRUGS —

Bitters before meals. —

℞ Sod Bicarb. gr x Spir Chloroformi ♏x
Tinct Nux Vom. ♏v Inf Gent Co ad ℥j
Inf Rhei ℥ij

* (Pot. Brom gr. x may be added)

Acids and ferments after meals (*see* p 385)

Note —Organic acids may and frequently do cause severe symptoms of 'hyperacidity'. In such cases avoid acids in initial stages, improving digestion by bitters, alkalis, and diet Then, later, give ferments and acids to replace the deficiency in the gastric juice

Carminatives —Essential oils, e g, of caiuput ♏ij in water, peppermint water, diluted with warm water, or creasote ♏j in capsules. Gentle abdominal massage

Atony and Dilatation.—*See* p 390.

Vomiting.—Usually allayed by treatment of dyspepsia. *If severe,* morphia or hydrocyanic acid, e g.

℞ ℞ Chloroformi
Acid. Hydrocyan. Dil. } āā ♏ij | Glycerin ℥ss
Liq. Morphin. Acetatis | Aq ad ℥ss

Lavage, very valuable.

Pain.—Alkalis and general treatment of dyspepsia. Special measures :—

(1) Spiritus ætheris (℥ss), with or without spiritus ammoniæ aromaticus (℥ss).

(2) Morphia, hydrocyanic acid, chloroform, and bromides. *See* VOMITING *above*, or following prescriptions: —

℞ Liq. Morphin. Hydrochlor. ♏xx
Spir. Ætheris ʒss | Spir. Ammon. Aromat. ʒss
Aq. ad ℥j

t.d.s.

℞ Sod. Brom. gr. vj
Bismuth. Oxycarb. gr. x | Acid. Hydrocyan. Dil. ♏nj
Aq. Menth. Pip. ad ʒss

t.d.s.

(3) *Lavage.*

Constipation.—Salines and aperient waters, senna pods, cascara. Avoid purges. Mercury pill occasionally.

V. VOMITING

Vomiting centre is in medulla oblongata, closely associated with respiratory centre.

Process of vomiting: 1st Stage: Profuse salivation and nausea; may be cold sweat. 2nd Stage. One or more deep inspirations followed by closure of glottis. Contraction of diaphragm and abdominal walls compresses stomach. 'Retching' results while cardia is closed. 3rd Stage: Ejection of vomitus. First stage may be absent, and entire process effortless.

PATH OF STIMULI.—Fauces and pharynx: by glossopharyngeal nerve. Stomach, by vagus. Certain poisons, e.g., antimony, apomorphine, act directly on vomiting centre.

Common Causes.—(1) Alimentary conditions: (a) Dyspepsia, gastritis, and gastro-enteritis of all types (*See* ACUTE GASTRITIS and DYSPEPSIA), (b) Acute peritonitis and intestinal obstruction, and acute abdominal disease, e.g., appendicitis. (c) Œsophageal obstruction. (2) Nephritis and uræmia. (3) Pregnancy. (4) Acute specific fevers; especially at onset. (5) Early phthisis. (6) Nervous system. (a) Cerebral disease—meningitis, tumour, abscess, etc.; (b) Gastric crises; (c) Hysteria; (d) Sea-sickness. (7) Eclampsia, diabetes, and conditions of acidosis.

Among other causes: (8) Drugs (emetics): zinc sulphate, ipecacuanha, apomorphine, antimony, etc. (9) Pain: biliary or renal colic, injuries to testes, etc.; also migraine. (10) Psychical: smells, sights, and emotions. (11) Reflex from fauces. (12) Mechanical vomiting, e.g., severe cough (compression of stomach). (13) Cyclical vomiting in children (associated with acidosis). 'sick headaches'. (14) Addison's disease. (15) Constipation.

SUDDEN ONSET.—Especially important: (a) Acute abdominal disease, e.g., appendicitis; (b) Acute specific fevers; (c) Toxic poisons.

VOMITING IN CHILDREN.—Acute specific fevers. Acute gastritis or gastro-enteritis, acute abdominal disease. Rarely: periodic or cyclical vomiting (with ketonuria).

Vomiting -Common Causes, *continued.*

TIME OF VOMITING --

EARLY MORNING --(1) Alcohol; (2) Pregnancy; (3) Renal. Occasionally from excessive salivation, e g, in pharyngeal catarrh

AFTER MEALS --Dyspepsia Gastric ulcer from pain during digestion (often constant interval one quarter to two hours). Neuroses (immediately after ingestion)

NO RELATION TO FOOD --Dilatation of stomach Cerebral disease Gastric cancer

Special Characters.

NAUSEA ABSENT -In cerebral conditions, gastric crises, and neuroses

BLOOD --Specially associated with or Gastric ulcer, or Cancer (See HAEMATEMESIS, p [illegible])

FECAL In acute intestinal obstruction after stages of great fecal accumulation Thin fluid Fecal masses very rare, and only in hysteria

NOTE -In intestinal obstruction vomit copious and fecal In peritonitis, vomit [illegible] markedly fecal

VI. CIRRHOSIS OF THE STOMACH

(Gastric Lipoma)

A chronic fibrosis of the stomach [illegible] causing the [illegible] and resulting in great thickening [illegible] with reduction of the lumen Rare

Etiology. Sex [illegible] Occurs [illegible] the muscularis [illegible] No relation to syphilis or to [illegible] Rare in [illegible] in certain

RELATION TO CANCER No [illegible] change [illegible] Cases are recorded with [illegible] and metastatic growths usually [illegible] Probably [illegible] and malignant

Ulcer occasionally present, and may be a predisposing cause

Morbid Anatomy. -

MACROSCOPIC -Stomach small smooth, heavy elastic Usually sausage shaped, holds only a few ounces No dilatation on opening On section Opaque white Thickening most at pylorus, may be an inch Usually limited by pylorus and cardia *All coats distinct.*

HISTOLOGY -Mucous membrane little change some small *round* cells Thickening due to fibrous tissue, well formed strands, mainly in *submucosa* and, to less degree, in muscular coats. Hypertrophy of muscular layers may be present

Changes are usually limited to stomach Sometimes colon, rectum, and ileum are affected Occasionally grades of chronic peritonitis with perigastric adhesions, etc., occur, merging into condition described under chronic proliferative peritonitis.

Symptoms.—Indefinite at onset: progress slow, may be years. *Vomiting*: At first occasional; later inability to retain even small amounts. *Epigastric pain*: Becoming continuous. *Tumour*: In epigastrium: smooth, firm, round or sausage-shaped, fairly movable; never dull. *Gastric contents*: Free HCl usually absent, not invariably. *Progressive asthenia*. Occasionally: hæmatemesis (but rarely copious); epigastric tenderness.

Diagnosis. Specially note: (1) Long duration; (2) Small capacity of stomach; (3) Character of tumour; (4) Opaque meal and X rays.

Treatment.—Exploratory operation. Go to enterostomy or gastrostomy if possible.

Hypertrophic Stenosis of Pylorus in Adults.—A condition occasionally recorded, is probably a localized type of cirrhosis of the stomach.

VII. ACUTE DILATATION OF THE STOMACH.

Occurrence.—

1. Post-operative, or during general anæsthesia. Accounts for [illegible] per cent of cases. Interval may be up to several days. Specially with but [illegible] operations on kidney and gall-bladder; also after stomach operations, even with gastro-enterostomy.

Rare:

2. In conditions of [illegible] especially wasting, e.g. chronic nephritis, diabetes.
3. Injuries to head or spine.
4. Lead poisoning.
5. Injuries to abdomen.

Symptoms. (1) Sudden onset. (2) Vomiting, enormous quantities, abdomen may be distended. (3) Collapse.

Mortality: [illegible] per cent. Diagnosis simple.

Etiology. Probably related to acute intestinal obstruction.

Duodenum always dilated as well as stomach. Dilatation may end at: (1) Level of superior mesenteric vessels crossing duodenum, constriction assumed to traction on root of mesentery owing to coils of gut prolapsing into pelvis, mesentery in these cases being abnormally long. (2) Below level of vessels, tentatively ascribed to prolapse of previously atonically dilated stomach, and kinking of the duodenum, or possibly pressure on duodenum by overdistended stomach.

Treatment.—(1) Pass stomach-tube and empty stomach repeatedly. (2) Turn on to abdomen and raise hips. (3) Stimulants; saline enemata.

Operation contra-indicated. Results very bad.

VIII.—CHRONIC DILATATION OF THE STOMACH.

(*Gastrectasis Atony of Stomach Motor Insufficiency*)

Causes.—(1) Atony of muscular coat of stomach loss of tone (2) Obstruction to exit of food: pyloric obstruction dilatation secondary

(1) ATONY OF STOMACH. ATONIC DILATATION.

ETIOLOGY.—(1) Constant overfilling with food or drink (2) Chronic catarrh, often with preceding e.g. beer drinkers (3) Poor general health and physique depression, neurosis especially in middle aged women: often with gastroptosis Uncommon under 40 years

Normal capacity of average stomach about 35 ounces maximum about 50 ounces over: pints without inconvenient distention is pathological

Dilatation results from muscular weakness affecting both tone and peristalsis When stomach is not emptied by peristalsis tone attempts to act continuously until exhaustion occurs and stomach commences to dilate The weaker muscle also results in feebler peristalsis, and work moreover is greater in raising contents to the pylorus Dilatation thus proceeds

SYMPTOMS.—Long duration and gradual onset

Dyspepsia.—Epigastric discomfort after food pain uncommon

Appetite.—Diminished or normal in latter, rapid satiety after small amounts (pressure rises rapidly in stomach lumen completely relaxed at onset

Flatulence.—Usually marked much fermentation

Vomiting.—*Uncommon*

General nutrition poor Skin dry and flabby tongue furred Teeth bad Constipation usually severe occasionally diarrhœa Palpitations Dyspnœa *Tetany* (q.v.) is a serious symptom.

PHYSICAL SIGNS.—

Inspection.—Abdomen prominent at umbilicus, depressed in epigastrium (Examine erect)

Curvatures of Stomach.—Both may be visible, (a) lesser below in form (b) greater below umbilicus

Peristalsis not visible

Artificial Inflation.—Tartaric acid ʒj. followed by sod bicarb. ʒiss, each in half tumbler of water For inspection and percussion of outline.

Palpation.—Splashing (*clapotage*) on bimanual examination or shaking patient: no value within two hours of meal may be auscultated.

Percussion.—Of little value except after inflation. Auscultatory percussion unreliable.

GASTRIC ANALYSIS: EWALD'S TEST MEAL.—(1) Free HCl: usually present, but nearly always diminished; occasionally high. (2) Total acidity: usually normal or increased, owing to increase of organic acids. Numerous sarcinæ and bacteria.

X RAYS: APPEARANCES AFTER OPAQUE MEAL.—

1. Opacity shaped like a bowl: upper level horizontal, lower crescentic.
2. Lower level several inches below umbilicus: may reach pubes.
3. Peristalsis practically absent.
4. Meal retained many hours (over 8), may be days.
5. Duodenal cap absent or ill defined.

TREATMENT.—General health of great importance: rest, exercise, fresh air, tonics. Teeth attended to. Bowels regulated: liquid paraffin, usually also aperients necessary, e.g. senna pods. Abdominal massage.

If much vomiting: Wet Mist belt treatment is preliminary.

LAVAGE.—Essential. Wash stomach once daily, warm solution of sod. chloride ℥j or sod. bicarb. ℥ij to pint: pour in two pints, siphon, repeat until fairly clear (about 5 pints). As improvement proceeds, reduce to once a week. (Patient can be taught to do this.)

MEALS.—At regular hours. *Rest 30 minutes before* and if possible one hour after, lying (also sleeping) on right side. Mastication slow.

DIET.—Amount normal, but easily digestible and stimulating to gastric juice. Meat extracts, minced meat (lightly cooked), fish, eggs, toast, rusks. Carbohydrates diminished, not in soft and pappy forms, but needing mastication, e.g. not mashed potatoes and milky foods. Fats avoided, except butter in moderation.

FLUIDS.—Meals taken dry. Fluids between meals. Hot water night and morning. At least 2½ pints of fluid daily.

DRUGS.—Bitters before meals. Acids and ferments after meals. (*See* HYPOCHLORHYDRIA.)

ABDOMINAL BELT may assist. Massage is advisable if a belt is worn.

GASTRO-JEJUNOSTOMY: unsatisfactory.

PROGNOSIS.—If lavage be continued once or twice weekly, and dietetic rules generally followed, comfort and fair health are obtained.

B. PYLORIC OBSTRUCTION OR STENOSIS.*

ETIOLOGY.—(1) Cicatrix of ulcer. (2) Neoplasm. (3) Less common: adhesions to gall-bladder, rarely chronic peritonitis. In children, congenital pyloric stenosis (q.v.).

Two types of stomach occur with pyloric obstruction: (1) Dilated stomach: atony. (2) Hypertrophied stomach of normal size: compensation maintained. Less common.

* Subject considered here for convenience.

Pyloric Obstruction, *continued.*

(1.) **Dilated Stomach from Pyloric Obstruction.**—Symptoms and physical signs and radiograph resemble primary atony of stomach (*see above*) with following exceptions:

SYMPTOMS.—May be previous history of peptic ulcer.

a VOMITING.—Characteristic symptom. Note (i) Quantity large. (ii) Intervals irregular—often several days—no direct relation to food. Gives temporary relief—then symptoms recur.

Characters of Vomit.—Sour smell. Separates into three layers, froth, fluid, and food—in lower level articles ingested long previously. Differs from vomit of primary atony (i) Bile absent. (ii) Often highly acid (Langdon Brown).

b. PAIN.—May be severe.

PHYSICAL SIGNS:

a. PERISTALSIS.—May be visible. Especially after food and with surface stimulation.

b. TUMOUR AT PYLORUS.—When present best felt on arrival of a peristaltic wave.

TREATMENT.—Operation—gastro-enterostomy—should be preliminary medical treatment as in primary atony.

(2.) **Hypertrophied Stomach from Pyloric Obstruction.** In this type the walls of the stomach maintain their tone and hypertrophy without dilatation.

SYMPTOMS.—May resemble ulcer.

a EPIGASTRIC DISCOMFORT AFTER FOOD.—Pain may be severe and spasmodic.

APPETITE.—May be normal but [illegible]

VOMITING.—May [illegible] not excessive.

PHYSICAL SIGNS:

INSPECTION.—*Peristalsis* [illegible] epigastrium, may be [illegible] prolapse of stomach.

PALPATION.—Splashing [illegible] palpable at pylorus.

X RAYS.—APPEARANCES AFTER OATMEAL MEAL.

a. Stomach practically normal in [illegible] umbilicus but meal accumulates in a bowl-shaped [illegible]

b. Peristalsis often occasional powerful wave.

c. Meal retained many hours—[illegible] may be days.

TREATMENT.—See previous type.

✓ IX. GASTRIC ULCER.

Loss of tissue in the mucous membrane and deeper coats of the stomach, characterized clinically by epigastric pain related to food, vomiting, and hæmatemesis. Ulcers may be acute or chronic, the incidence and other factors varying considerably in the two forms.

ETIOLOGY.

AGE, SEX (1) *Acute ulcers*: Great majority in young females, age 20 to 30 years, especially anæmic girls. (2) *Chronic ulcer*: Equally common in males and females, age over 30 years.

OCCUPATION: Especially in young servants. Also in cooks and among men bakers.

DIET: Influence unknown but probably exists.

Alcohol, trauma, heredity: no obvious influence. Syphilis, tubercle: no influence.

The cause of production of peptic ulcers is unknown. Experimental gastric ulcers in animals heal readily unless acidity of gastric juice is artificially raised. Especially the gastric mucous membrane is subject to abrasion due to [illegible] which normally heal readily, but lead to ulcers in presence of hyperacidity. Gastric motility may be [illegible] used in gastric and hyperacidity [illegible]. Acute gastric ulcers usually heal [illegible] unknown why they sometimes become chronic.

MORBID ANATOMY AND GENERAL DESCRIPTION.

ACUTE ULCER: Not uncommonly multiple, nearly half [illegible] position, more frequently [illegible] curvature [illegible] than posterior wall. Size [illegible] a pea to a halfpenny. [illegible] affected than the mucosa, [illegible] or surrounding inflammation [illegible]. Age [illegible] oedema [illegible] greyish appearance [illegible] peritoneal [illegible]. Hæmorrhage rarely fatal. Perforation [illegible] in general peritonitis.

CHRONIC ULCER: Rarely multiple. [illegible] lesser curvature, posterior surface [illegible]. May cover several inches. Edges are hard, [illegible] contraction [illegible]. Wall [illegible] and indurated. Base smooth or scarred, formed by deeper layers of the [illegible] organs, e.g. pancreas. Inflammatory changes in [illegible]. Hæmorrhage not uncommonly fatal. Perforation less common than in acute ulcer and abscess may be localised.

MODE OF HEALING: Granulation tissue spreads in from edge. Acute ulcers heal with little scarring or sequelæ. Chronic ulcers after years may show no healing in one part while scarred in another. Edges of large ulcer may produce serious results: (1) Pyloric stenosis. (2) Hourglass stomach, from saddle-ulcer involving anterior surface.

EROSIONS: Small abrasions or slits in the mucosa, usually multiple. Occasionally cause severe hæmorrhage (Hale White's "gastrostaxis").

Gastric Ulcer—Erosions, *continued.*

Occur rarely in new-born infants, wasting conditions of children or adults (e.g., cirrhosis of liver), and in septicæmia or pneumococcal affections.

Pathological Effects produced by an Ulcer.—(1) Perforation; (2) Erosion of blood-vessels; (3) Cicatrization—(a) pyloric stenosis, (b) hour-glass stomach; (4) Perigastric adhesions; (5) Cancer. Very rarely, general subcutaneous emphysema (probably anaerobic bacilli).

PERFORATION.—*Site*: on anterior wall in 70 per cent. More frequent in acute ulcers (from mobility of stomach and absence of adhesions); hence total incidence greater in women, but of perforated chronic ulcers, over age of 30, more occur in men. May be multiple. *Results* depend on site, size of perforation, and adhesions: (1) Generalized peritonitis, especially from anterior wall and acute ulcers; (2) Localized abscess—e.g., chronic ulcer ruptures into lesser sac, and produces subphrenic abscess. (3) Very rarely, perforates into adherent intestine, usually into the transverse colon. Very rarely into pleura or pericardium.

EROSION OF BLOOD-VESSELS: HÆMORRHAGE.—Frequent both in acute and chronic ulcers, more often fatal in latter from exposure of deeper and larger arteries. Commonest on lesser curvature, from branches of coronary or gastro-epiploic arteries; in chronic ulcers often on posterior surface, especially from the splenic artery. Changes in the vessels, embolism, endarteritis, or small aneurysms occasionally present.

CICATRIZATION.—Acute ulcers on healing usually leave small, harmless scars. From chronic ulcers, serious results are:—

PYLORIC STENOSIS.—Dilatation of stomach may result. Pyloric obstruction caused by: (a) Scarring, chiefly; (b) Spasm near ulcer; (c) Adhesions and kinking of duodenum.

HOUR-GLASS CONTRACTION.—Ulcer usually on lesser curvature involving anterior surface; constriction divides stomach into two pouches; orifice may admit a pencil only. Occasionally pyloric stenosis is also present.

ADHESIONS TO OTHER ORGANS.

PERIGASTRIC ADHESIONS.—In chronic ulcers very frequent, especially on posterior surface or near pylorus; tend to prevent healing, but diminish risk of perforative peritonitis.

VISCERA INVOLVED.—(1) Pancreas, 50 per cent of adhesions. (2) Liver, 25 per cent; (3) Less often colon, spleen, mesentery, etc. (Gastric adhesions also occur in disease of gall bladder, pancreas, syphilitic liver, and may be extensive in chronic peritonitis.)

RESULTS OF ADHESIONS.—May be impaired motility, pyloric obstruction, hypertrophy of stomach. Rarely, chronic plastic peritonitis present.

SYMPTOMS.—(1) Pain: frequent, influenced by posture, relieved on lying down, increased by pressure, less affected by diet and less intermittent than in gastric ulcer. (2) Local

tenderness, may be tumour near pylorus. (3) General condition good. *Dilated stomach rare.*

X RAYS: APPEARANCE AFTER OPAQUE MEAL.—(1) 'Shadow defects'; (2) Interference with peristaltic waves. (*See* CANCER OF THE STOMACH.)

CARCINOMA SECONDARY TO GASTRIC ULCER. *See* p. 494.

SYMPTOMS.

Characteristic Symptoms.—(1) Pain in epigastrium definitely related to food; (2) Vomiting; (3) Hæmatemesis; (4) Increased total acidity and free HCl in gastric contents.

MODE OF ONSET.—Types:—

1. Latent. First symptom hæmatemesis or even perforation, especially in acute ulcers.
2. Dyspepsia may exist for years before definite symptoms.
3. Definite symptoms occur early.

PAIN.—Rarely absent.

SITE.—*Epigastrium*, frequently just below ensiform: usually localized. Also frequently in back, at tenth dorsal vertebra: pain may shoot through, or spread round left side. In chronic ulcers, often lower in epigastrium and more diffuse.

FOLLOWS OR AGGRAVATED BY FOOD.—Recurring regularly one-quarter to two hours after meal. Rapid onset suggests ulcer at cardia, but interval may be brief with ulcer at pylorus. Rarely, at night or with empty stomach (? due to continuous HCl secretion).

DURATION.—Varies: several hours, often until passage through pylorus or vomiting empties stomach. Is not continuous, though in severe cases discomfort may be persistent. In early stages not severe. May be burning or hungry; or in severe paroxysms. May be freedom for weeks on end; then recurrence. Increased by pressure, even 'the weight of the clothes'.

SUPERFICIAL TENDERNESS.—Small area, ½ to 1 inch sharply defined: usually between ensiform and left costal margin. Not always present. Less often similar dorsal area between 7th and 11th dorsal spine, slightly to left.

CAUSE OF PAIN AND HYPERÆSTHESIA OF SKIN.—Is reflected pain, a visceral cutaneous reflex. Tenderness accompanied by, and possibly due to, localized spasm of rectus; doubtful if stomach itself is tender. Origin of pain uncertain: perhaps from gastric distention or abnormality of peristalsis; not due to direct irritation of nerves in ulcer.

SITE OF ULCER AND RELATION TO PAIN AND TENDERNESS.—No reliable evidence as to site is afforded by position of pain and tenderness, or by time of occurrence after food (except ulcers immediately affecting cardiac orifice).

VOMITING.—Common but not invariable. Usually at height of pain, giving relief. Bile rare.

HÆMATEMESIS.—In about one-third. *Premonitory*: oppression in stomach, faintness. Blood brought up without any effort.

Gastric Ulcer—Symptoms, *continued*

Subsequently, cold sweat, pallor, rapid pulse, fainting. May be several attacks. *Vomit large,* blood airless, characteristically 'coffee grounds' appearance.

Results. (2) Anæmia, symptoms of ulcer often temporarily absent, slight fever. (3) Death mostly in chronic ulcers involving splenic artery. Rare. (4) Amaurosis. (1) Convulsions, if from cerebral anæmia, recovery, but if from thrombosis permanent hemiplegia. (5) Melæna occurs occasionally if ulcer near pylorus, rarely without hæmatemesis.

GASTRIC CONTENTS. Acidity both total and free HCl is increased. With Ewald's test meal, average total acidity 50 to 100, decinormal HCl per cent (equal to HCl 0·20 to 0·40 gram per cent) and free HCl 35 (equals 0·13 gram per cent). (Normal total acidity is 40 to 50, free HCl 22 to 33 (equals 0·08 to 0·12).

Other and less Characteristic Symptoms.

GASTRO-INTESTINAL—

APPETITE. Often good, but 'afraid to eat.'

TASTE. Frequently carious but may be very good.

DYSPEPSIA. Flatulence, etc., common, especially in chronic ulcers. Severity of all kinds.

CONSTIPATION. Rarely absent.

ANÆMIA. Of secondary type, hæmoglobin favourable to red cells, and hence colour index low. Of all degrees, depending on chronicity, hæmatemesis, and diet.

WASTING. Present but not extreme. In severe cases patient is pale and flabby. In chronic ulcers may be slight.

Physical Signs.—Search for upper abdominal and lower epigastrium and for pyloric obstruction and dilatation.

X Rays: Appearances after Opaque Meal.

(1) ULCER OF FUNDUS—

a. SPASMODIC CONTRACTIONS. A long persisting [illegible] strong evidence of [illegible]. May simulate [illegible] stomach, differentiated by (i) relaxation on [illegible] massage, (ii) repeat examination. With a [illegible] contraction will be towards any point of [illegible] to pass across [illegible] lesser curvature across shadow be on right.

b. Bismuth may be retained in lumen of chronic penetrating ulcers.

(2) ULCER AT PYLORUS. In addition to above note:

(1) SHADOW OF PYLORUS AND DUODENUM [illegible].

(2) STOMACH EMPTIES SLOWLY, i.e., over 6 hours. May also occur with ulcer of fundus.

COMPLICATIONS AND SEQUELÆ.

SUMMARY.—(1) Hæmatemesis (p. 400). (2) Perforation. (3) Anæmia. (4) Pyloric stenosis and dilatation of the stomach (p. 390). (5) Hour-glass stomach. (6) Perigastric adhesions. (7) Jejunal ulcer. (*See* MORBID ANATOMY.)

Perforation of Gastric Ulcer.—Especially acute ulcers on anterior wall and near pylorus, causing general peritonitis. Localized or subphrenic abscess from chronic ulcers on posterior wall.

SYMPTOMS.—Sudden onset. Severe continuous pain, commences in epigastrium, spreads over abdomen, rarely in right iliac fossa. Vomiting not invariable. Temperature, early subnormal, then rises. Shock: variable. Early, pulse often strong, but rate increases steadily.

PHYSICAL SIGNS.—Abdomen rigid, tender, and in early stages retracted. Liver dullness often absent in early stage; valuable sign of free gas, in later distended stages is simulated by distended coils.

LATER STAGES.—Condition of general peritonitis.

LATENT PERIOD.—Immediate symptoms of perforation may subside in half to one hour, and apparent follows in which signs or symptoms are present. Duration few hours. Then general peritonitis develops.

PROGNOSIS.—Often die in a few hours.

TREATMENT.—Immediate operation.

LOCAL OR SUBPHRENIC ABSCESS.—Intermittent symptoms as above. Temporary improvement. Symptoms of suppuration. Abscess may give physical signs. [illegible] Abscess.

Hour-glass Stomach. Characteristic symptoms. Manifest: (1) On washing out some fluid lost. (2) After emptying and washing stomach gastric contents reappear sooner or later. (3) After emptying stomach, splashing present in [illegible] dilatation. (4) After distention with [illegible] position, sizzling [illegible] visible, rarely two tumours visible. (5) X rays and opaque meal (p. 396).

TREATMENT.—Gastroenterostomy to [illegible] operation. If [illegible] stenosis also present, gastroenterostomy to [illegible] pylorus.

Jejunal Ulcer. A sequel of gastro-enterostomy, especially by anterior method, opening between [illegible] contents to unsterilized cicatrix; rare in posterior method or recent technique.

SITE.—Often at anastomosis, in efferent limb, rarely in afferent.

SYMPTOMS.—Tenderness and pain in epigastrium, relation to food varies; food may give relief. Onset often at night. Occasionally perforates or bleeds.

PREVENTION.—Posterior method, and alkalis after operation.

DIAGNOSIS.

Simple, with characteristic symptoms, viz.: (1) Pain localized, recurring regularly, following or aggravated by food; (2) Tenderness; (3) Cutaneous hyperæsthesia over small area. (4) Hæmateneses; (5) Vomiting, easing pain usually. (6) Gastric hyperacidity. Also X rays and opaque meals.

ACUTE ULCERS (young women).—Diagnosis from:—

(1) Chlorosis.—Dyspepsia common, pain not localized, nor following food; great improvement with iron.

Gastric Ulcer—Diagnosis, *continued.*

2. HYPERCHLORHYDRIA.—Relieved by alkalis and diet. (Relation to ulcer uncertain.)

CHRONIC ULCERS —Diagnosis from :—

1. CHRONIC GASTRITIS.—Pain not localized, vomiting irregular Gastric contents: acidity usually diminished
2. GASTRIC CRISES (TABES) - Pain and vomiting independent of food. Larger area of cutaneous hyperæsthesia (may precede loss of knee jerks and Argyll Robertson pupil)
3. CANCER.—Pain more continuous; rapid wasting: may be tumour. Gastric contents: (a) If short history, free HCl absent (important), (b) If long history, may be cancer following ulcer, free HCl present (no assistance in diagnosis (*See* CANCER OF THE STOMACH)
4. GALL-BLADDER DISEASE -Radiation of pain Gastric analysis free HCl usually lessened or absent Jaundice
5. DUODENAL ULCER.—Pain relieved by food, vomiting slight
6. MOVABLE KIDNEY Often palpable.

TREATMENT.

May be medical or surgical.

INDICATIONS FOR MEDICAL TREATMENT – (a) *Acute ulcers* especially under age of 30 years. (b) *Chronic ulcers* course of medical treatment should precede operation

NOTE *Hæmatemesis in young women*, operation contra indicated. bleeding points, often multiple (gastrostaxis), persist after gastrojejunostomy, This condition does well on medical treatment, and is rarely fatal

INDICATIONS FOR SURGICAL TREATMENT –

1. Perforation -Immediate operation
2. Hæmatemesis in chronic ulcers Delay not exceeding day or two
3. Conditions hindering passage of gastric contents Pyloric obstruction, hour glass stomach, perigastric adhesions
4. Chronic ulcers resisting medical treatment, especially over age of 40 years.

Medical Treatment.—Acute ulcer, at least four weeks. chronic ulcer, eight to ten weeks.

(1) WITHOUT RECENT HÆMATEMESIS *Indications* Rest to stomach and establishment of conditions to enable ulcer to heal But general weakness results if food by the mouth withheld long

1. REST IN BED —At least four weeks: not up until on full diet
2. DIET.—

(a) *Lenhartz's Method.*—Commences at once with milk and eggs, the diet being nutritious and also neutralizing acidity. proceed according to a definite scheme, increasing the number of eggs, and early adding raw beef and raw ham. Lenhartz's treatment includes: absolute rest at least four weeks, icebag on epigastrium two weeks, bismuth subnitrate gr. xxx, t.d.s for ten days, bowels opened with

enemata commencing in second week. Results very good. (Lenhartz's original scheme is often modified, and duration is frequently too short.)

b. *Stricter Diet.*—Preferred by many authorities. Approximately—First week: *milk*, commencing one pint in twenty four hours in small frequent quantities, amount gradually increasing. Second week: milk, bread and butter, eggs. Third week: chicken. Fourth week: mixed diet. Diet always strictly regulated: if pain occurs, return to early stage of diet: if hæmatemesis, treat as below.

3. DRUGS.—As regards the ulcer, all drugs useless except bismuth and alkalis action neutralizing acid and not protective to ulcer.

℞ Bismuth. Oxycarb. } ãã
Mag. Carb. Pond. } gr. xx
Sod. Bicarb.
7 or 7ss t.d.s.

4. GENERAL TREATMENT:—

Mouth. *Remove all septic teeth*, cleanse mouth carefully.

Bowels.—Regulate with enemata, or with saline aperients.

Anæmia. Blaud's pills, commencing in third week.

Ice bag to epigastrium eases pain and allays flatulence.

5. COMPLICATIONS.—

Pain. Usually subsides with rest, ice bag to epigastrium, and bismuth. If severe, add opium (gr. ¼) to bismuth powder; avoid hypodermic injection. Warm fomentations may relieve.

Vomiting and Irritation of Stomach.—If not subsiding under treatment, wash out stomach gently with weak alkali daily (sod. bicarb. ʒj to pint).

WITH RECENT HÆMATEMESIS.—Absolute rest, flat in bed. Hypodermic injection of morphia gr. ¼, immediately.

1. DIET.—Practice varies:—

a. *Lenhartz.*—Commences his treatment at once. Pain, acidity, and irritability of stomach are generally less after hæmatemesis. The treatment is well borne and results are good.

b. *Enemata.*—Nothing by mouth, or, at most, sips of water. Feeding by nutrient enemata: the period recommended varies, three to ten days. Then commence milk.

Nutrient Enemata.—Numerous investigations prove no absorption of protein from rectum. Only substances of value are water, glucose, and alcohol. Enema of maximum value is: normal physiological saline containing 5 per cent glucose, 2 pints, with brandy 3 ounces.

Note two precautions in treatment with enemata: (a) Cleanse mouth, to avoid parotitis; (b) Examine urine for acidosis.

Gastric Ulcer—Medical Treatment, *continued*

Treatment recommended Leubart's method If hematemesis returns, or stomach very irritable, enemata as above, t d s for two to three days then Leubart's diet

2 Drugs Avoid all except bismuth Following especially have been suggested may be tried in repeated hematemesis of young women in order- adrenalin chloride (1 1000) ℥ x to xx t d s in a little water, oil of turpentine ℥ t d s (℥) beaten up in white of one egg

SUBSEQUENT TREATMENT with or without operation Should be on full diet before setting up, and continue unchanged for several weeks *after discharge* Meals regular keep bowels regular liquid paraffin good Course of bismuth and alkalis occasionally

Prognosis.

ACUTE ULCER —Medical treatment often insufficiently prolonged and ulcer recurs Ulcer treated with medical treatment prognosis very good Chronic ulcer apparently always usually result from ulcer thus treated

CHRONIC ULCER Greater risk of fatal hemorrhage also of cicatrization of intestine and cancer Gastric pain very good very good results may obtain in [illegible] Medical treatment of pyloric ulcer usually gives permanent relief but ulcer usually recurs

X. DUODENAL ULCER.

Loss of tissue in the mucous and deeper layers of the duodenum characterized clinically by epigastric pain, vomiting blood, by melena, and by high gastric acidity

Etiology and Morbid Anatomy. -

SEX —Great majority in males

AGE —Usually over 30 years Rare in childhood [illegible] in marasmic infants

SITUATION OF ULCER In first part of duodenum in [illegible] per cent, usually within two inches of pylorus [illegible] papilla Generally on upper portion of anterior wall

Occasionally difficult to decide If ulcer is pyloric or duodenal Mayo gives pyloric vein as line of demarcation

NUMBER —Usually single Multiple rare

BURNS —Rare sequel of burns, usually extensive Very rarely in other forms of sepsis. Cause unknown possibly septic emboli

Complications and Sequelæ. - Resemble those in gastric ulcer, except that carcinoma is extremely rare

EROSION OF BLOOD-VESSELS Especially in ulcers on posterior wall, owing to position of arteries Usual vessels eroded superior pancreaticoduodenal and gastroduodenal arteries

PERFORATION —Especially with ulcers on anterior wall General peritonitis results from infection of general peritoneal cavity

localized abscess less common. Symptoms as in perforated gastric ulcer, but with pain in right epigastrium. May simulate perforation of appendix.

CICATRIZATION.—Ulcers near *pylorus* may cause obstruction and dilatation of stomach. Severe scarring less common than in gastric ulcer.

ADHESIONS.—To liver, gall bladder, or pancreas.

Symptoms.—

INITIAL SYMPTOMS.—(1) Latent—initial symptom perforation or serious hæmorrhage or scar found post mortem. (2) Indefinite dyspepsia. (3) Chronic course like symptoms. (4) Dilatation of stomach.

CHARACTERISTIC SYMPTOMS.—(1) Pain relieved by food. (2) Melæna. (3) Increased gastric acidity. Attacks frequently intermittent but extending over many years.

PAIN.—Severe. *Usually comes* [illegible] two hours after food, or often at night [illegible] probably due to closure of pylorus and protection of ulcer from acid gastric juice. Not eased by vomiting. Site: In right of epigastrium, and above umbilicus. Radiates to epigastrium, umbilicus, and right side, never to subscapular region (as in hepatic pain). Definite localization on [illegible]. May be rigidity of right rectus, and area of superficial tenderness. Occasionally the pain is in centre or even to left of epigastrium. Is possibly due to pylorospasm.

HÆMORRHAGE.—Melæna [illegible] often repeated. Rapid fatality rare. On [illegible] faintness, cold sweat and rapid pulse [illegible]. On passing of melæna, sudden colicky pain, sudden call to stool, and [illegible] tarry [illegible]. Both the melæna and [illegible] of bleeding may be unrecognized by patient.

Occult blood.—Fresh in fæces may be shown by tests for "occult blood."

Hæmatemesis may also occur, depending on site of ulcer, rarely without melæna.

GASTRIC CONTENTS.—*Hyperacid.*—Acidity generally very high. With Ewald's test meal, mean total acidity 70 equivalent HCl 0.22, free HCl 45 equivalent HCl 0.15. Higher acidities are common. (*See* Gastric Ulcer, p. 396.)

DYSPEPSIA.—Variable. Chronic dyspepsia may precede perforation or hæmorrhage. Appetite often normal. Flatulence and heartburn frequent. *Vomiting uncommon.*

ANÆMIA.—May be [illegible] increase, from repeated hæmorrhages.

WASTING.—Usually slight. Nutrition often very good.

ORAL SEPSIS.—Common.

X Rays: Appearances after Opaque Meal.—

1. Stomach normal in shape, but peristalsis active and organ empties very rapidly, often in one hour.
2. Duodenal cap irregular in outline.
3. Food may pass through duodenum in large masses.

Duodenal Ulcer, *continued*

Diagnosis.—Simple if characteristic symptoms present. Often difficult. From —

GALL-BLADDER DISEASES.—Pain radiates to right shoulder; no melæna; gastric contents free HCl usually absent. Jaundice.

GASTRIC ULCER.—Character of pain; frequency of vomiting. Duodenal ulcer very rare in young women.

GASTRIC CRISES (TABES).—Pain and vomiting independent of food. Larger area of cutaneous hyperæsthesia.

Note. Gastric crises may occur before loss of knee jerks and Argyll Robertson pupil.

MOVABLE KIDNEY.—Generally palpable; no hæmorrhage.

APPENDICITIS.—After perforation of a duodenal ulcer, pus may track down into right iliac fossa, and closely simulate perforation of appendix.

Treatment.—

MEDICAL TREATMENT.—*In a short history and no evidence of hæmorrhage,* medical treatment should be carried out as in gastric ulcer, the essentials being rest and prolonged treatment on a definite scheme. With such provision, results satisfactory.

SURGICAL TREATMENT.—Indicated for all other cases, and if symptoms return. Treatment after discharge should be carried out with same care as if operation had not been performed.

HÆMORRHAGE.—Operate within two days if severe.

Note.—If operation be not indicated, symptoms can usually be allayed by careful dieting without meat, some food in units, milk between chief meals, and alkalis; this procedure should not be forced, and the risks should be made clear.

Prognosis. Good, if under observation and treatment. Mortality from severe hæmorrhage high, and consequently operation must not be delayed if symptoms persist.

XI. CANCER OF THE STOMACH.

Etiology.—

INCIDENCE.—In males, most frequent form of cancer. In females, less common than uterus and breast. Accounts for 20 to 40 per cent of cases of cancer, and 1·5 to 2 per cent of all deaths.

SEX.—More frequent in males. Statistics vary, possibly 2 to 1.

AGE.—Commonest between 40 and 60 years. Rare under 30 years.

IRRITATION.—Predominance at certain sites ascribed to exposure to local irritation.

RELATION TO GASTRIC ULCER.—*See p. 404.*

External trauma, alcohol, tuberculosis of no influence. Heredity, data very incomplete, no proof of influence.

General Features of Morbid Anatomy.—

FREQUENCY AT DIFFERENT SITES.—(1) Pylorus, 60 per cent. (2) Lesser curvature, 10 to 15 per cent. (3) Cardia, 8 to 10

per cent. (4) Posterior wall, 5 per cent; (5) Whole or extensive, 5 per cent. Anterior wall, greater curvature, fundus, rare.

MACROSCOPIC APPEARANCE.—(a) Frequently an ulcer with rough floor and hard, irregular, everted edges, wall thick and adherent. (b) May be fungating masses. Hard and soft areas often coexist.

Early malignant ulcer distinguished from innocent often by microscopic sections only.

Spreads in submucous coat: on section the white translucent growth shows against dark hypertrophied muscle, may be ½ inch thick. Spread is by lymphatics.

PYLORIC CANCER. Characteristics. (1) Walls thickened; (2) Opening narrowed. (3) *Pylorus also everted into duodenum.* (4) *Duodenum everted*; usually scirrhous. Tends to spread along lesser curvature.

HOURGLASS STOMACH. [illegible] the thickened [illegible] scirrhus with much fibrous tissue and few cells; cancer [illegible] not yet excluded.

Morbid Histology. Four types of [illegible]: (i) Scirrhous, [illegible] per cent; (ii) Medullary; (iii) Colloid; (iv) Colloid degeneration and [illegible] common. May be grouped as—

1. SCIRRHOUS ALVEOLAR CARCINOMA.
   a. SCIRRHOUS. Common at pylorus. Very hard whitish little juice on cut surface. *Histology*: much stroma, few cells.
   b. MEDULLARY OR MEDULLARY. Soft masses, grayish white, much juice. Ulceration and [illegible] common. May coexist with scirrhous. *Histology*: masses of cell strands scanty.
2. COLUMNAR CELLED ADENOCARCINOMA. Large masses, moderately firm. [illegible] common and [illegible] ally [illegible] often [illegible] ulcer. *Histology*: not infrequent. Tendency to secondary growths in liver, lung, and [illegible].
3. COLLOID CARCINOMA. [illegible] as common. Spreads widely. *Often extends* to omentum and neighbouring organs. Forms large masses. *Histology*: alveoli very distinct, containing gelatinous colloid material and often a few large epithelial cells. Substance *differs from that of gelatin.*

Resultant Changes in the Stomach. Vary with [illegible] of tumour. (1) Cardia: stomach small, oesophagus dilated. (2) Pylorus: stomach usually dilated (*see* PYLORIC OBSTRUCTION, p. 491). (3) Body: little change.

Adhesions common, especially to pancreas, liver, and colon. In absence of adhesions, stomach often very mobile, owing to weight of tumour.

Secondary Growths.—Very common; in over 80 per cent at death.

LYMPHATIC GLANDS.—In 35 per cent at least. (1) Abdominal, frequently. (2) *Cervical*, occasionally: special gland at outer

Cancer of the Stomach—Secondary Growth, *continued.*

border of left sternomastoid infected by spread along thoracic duct; *great diagnostic importance* (3) Axillary, left Occasionally inguinal, etc

LIVER In about 30 per cent Often very large

OMENTUM, PERITONEUM, INTESTINE.—In 20 per cent Less common Pancreas, lungs, and pleura Occasionally Bones, brain, spleen other parts

SUBCUTANEOUS NODULES At or near navel peritoneum not necessarily affected

Secondary Neoplasms in Stomach.—Very rare Breast commonest primary site.

Carcinoma Secondary to Gastric Ulcer.—History suggesting previous ulcer present in 5 to 10 per cent usually long duration twenty to thirty years, gastric contents in these also contain increase of free HCl in total acidity Pathological examination and results of autopsy also suggest previous simple ulcer with similar frequency This is most probable percentage but Mayo and some others give up to 50 per cent the latter figure undoubtedly greatly excessive

Frequency of gastric ulcer becoming cancer is about 1 per cent At the margins of a chronic ulcer areas of regenerative may occur due to chronic inflammation liable to be mistaken histologically for malignancy

Carcinoma secondary to gastric ulcer has certain differences from the primary carcinoma described in paragraphs below

(1) MODE OF ONSET Two types, readily recognized with difficulty (a) Prolonged history of dyspepsia very rare, under 20 years, no previous history of ulcer (b) Short recent period of trouble, marked by definite exacerbation of symptoms developing conditions as in primary forms

(2) GASTRIC ANALYSIS (Fasting test meal) Free HCl present amount and total acidity normal or increased resembling gastric ulcer (Exceptions possible as in rarely but chronic gastritis)

DIAGNOSIS of carcinoma and differentiation from gastric ulcer rest principally on recognition of second stage

Symptoms.—

MODE OF ONSET —Gradual and insidious but history of gastric symptoms in *short, not exceeding six months, and previous dyspepsia strikingly rare. Initial complaint* usually pain, dyspepsia vomiting, or loss of weight Rarely, onset sudden

CARDINAL SYMPTOMS —

ANOREXIA —Especially for meat. Flatulence common

PAIN.—Early symptom; usually epigastric, may be referred to shoulder or back, worse after food, increased by pressure, partly, but not entirely, relieved by vomiting *Character* dragging, less intermittent than in gastric ulcer, and paroxysms, late. In cancer of cardia, dysphagia. *Superficial*

tenderness: usually small area (diameter 1 inch) anteriorly between nipple and umbilicus, and posteriorly between 5th and 12th dorsal spines, viz., 6th to 9th dorsal segments.

NAUSEA AND VOMITING.—At onset, occasional vomiting, often rapidly becoming more frequent. With cancer of cardia, shortly after food; with pyloric cancer, after an interval; with cancer of body, may be absent. Relief after vomiting in early stages; later very slight. Nausea becomes continuous.

LOSS OF WEIGHT.—Progressive, becoming extreme; factors are the growth, low diet, vomiting, and imperfect gastric juice. Temporary improvement may follow (1) dieting, (2) lavage (if stomach dilated), and may be deceptive. Loss of strength is proportional to loss of weight.

CACHECTIC PALLOR.—Often almost diagnostic; develops rapidly. Anæmia.

LESS CONSTANT AND IMPORTANT.

Hæmatemesis.—Rare; profuse, may be from ulceration of growth, causing "coffee-grounds" vomit; occasionally plentiful, arterial (? arterial blood).

Fever.—Variable; occasional, is not uncommon.

Œdema of ankles, and general results of anæmia. Urine: occasionally albuminuria; rarely acetonuria (starvation). Constipation, usual; traces of ascites.

Symptoms of [illegible] may [illegible] attention first, e.g. liver, [illegible].

LATENT CARCINOMA.—Often recognized only clinically or post mortem, without symptoms or signs.

Physical Signs.—Abdominal examination may be negative. If necessary, examine under general anæsthesia. Physical signs of gastric obstruction and dilatation [illegible].

INSPECTION.—Look for epigastric prominence, aortic pulsation, marked peristalsis, dilated stomach, subcutaneous nodules near navel; tumour may be visible, moving with respiration.

PALPATION.—Tumour frequently palpable; large, often lobular; mobility usually marked (4 or 4 in. best), moving with respiration; aortic pulsation, manipulation, and occasionally with peristalsis of stomach and also after inflation of stomach.

INFLATION OF STOMACH.—Outlines stomach and exhibits dilatation; often reveals peristalsis, and occasionally a tumour (Aviol with hæmatemesis). After inflation tumour of pylorus may move towards right of posterior wall, disappears.

NOTE ON TUMOUR.—Healthy pylorus is not palpable even after inflation. Tumour of pylorus often very mobile; but at cardia liver may cover it, more often impalpable; on greater curvature may resemble rolled omentum, colon tumour, or fæces. *Adhesions may later fix tumour*, e.g. on anterior wall, but adhesions often stretch. Tumour may be palpable one day and not so the next.

LYMPHATIC GLANDS.—Especially in neck, and left axilla.

LEATHER-BOTTLE OR INDIARUBBER-BOTTLE STOMACH.—Small hard mass, no increase on inflation, vomitus small in amount.

Cancer of the Stomach –**Physical Signs** *continued*

X RAYS: APPEARANCES AFTER OPAQUE MEAL –

1. Shadow defects: irregularities due to the growth
2. Interference with progress of peristaltic waves

Note – An indurated ulcer or perigastric adhesions may produce similar results and be indistinguishable

Gastric Analysis: Ewald's Test Meal.– Great importance in diagnosis. *Changes in gastric secretion are present at most early symptoms* and progressive diminution to final absence of free HCl is not observed. Characteristics: (1) Free HCl absent (Günzburg test). (2) Total acidity low, usually equivalent to 15 to 35 c.c. decinormal NaOH per 100 c.c. to 0.1 gm. HCl. Free acid lactic acid may be present, due to organic acid.

Note: (1) Increase of lactic and other organic acids may be serious and Oppler-Boas bacillus but of little diagnostic importance. (2) Blood hardly ever seen in gastric juice, extremely rare even in malignant gastric ulcer. (3) In pernicious anemia and achylia gastrica total acidity usually much lower and no free acids or ferments present.

Changes in the Blood. Anemia constant, usually type of hemoglobin reduced more than red cells and hence color index low. Changes of blood with inflammatory and cancer not uncommon.

When anemia marked, a few normoblasts may be present. In very rare cases [illegible] but leucocytosis and [illegible] blood may be present [illegible] Pernicious Anemia.

Complications.

(1) SECONDARY GROWTHS: Most [illegible] Anatomy.

(2) JAUNDICE: from [illegible] pressure [illegible] portal.

(3) PERFORATION: four [illegible] peritonitis or [illegible] colon, rarely into pleura or [illegible].

Occasionally: (4) Fatal hematemesis (spleen [illegible]); (5) Gangrene of growth; (6) Abscess or [illegible] saphena veins.

Course and Termination. Symptoms usually well marked in two to four months from initial trouble. *Progress* with rapid wasting, [illegible] and vomiting. *Duration* usually nine to eighteen months, rarely exceeds two years. *Death* from asthenia, with consciousness to the end, rarely in coma.

Diagnosis.—*Characteristics are* (1) Quick and rapid progress of dyspepsia and wasting *without previous gastric trouble*. (2) Absence

of free HCl; (3) Presence of tumour. (*See also* CARCINOMA SECONDARY TO GASTRIC ULCER, p. 404.)

METHODS OF DIAGNOSIS.—Include: (1) Symptoms and examination, under general anæsthetic if necessary; (2) Analysis of gastric contents; (3) Examination of blood; (4) X rays and opaque meal; (5) Occult blood in fæces; (6) Exploratory laparotomy in all cases of doubt.

Note.—(1) Absence of gastric symptoms practically excludes gastric cancer even when tumour present. (2) Since duration of symptoms of primary carcinoma does not exceed 2 years, and of carcinoma secondary to gastric ulcer is rarely under 20 years, symptoms of intermediate duration are very rarely due to carcinoma. (3) Absence of HCl with duration of symptoms exceeding 2 years is against carcinoma.

TUMOUR NOT PALPABLE. Difficulties are:—

CHRONIC GASTRITIS.—*Long gastric symptoms with absence of free HCl; cachexia.*

DILATED STOMACH.—Long gastric symptoms. X rays.

GASTRIC ULCER.—Increased gastric acidity; also pain worse after food, relieved by vomiting, waxing less; hæmatemesis more frequent, duration longer.

PERNICIOUS ANÆMIA.—Changes in the blood; almost total absence of gastric secretion, total acidity often under 10. Rare difficulties in secondary deposits of cancer in long bones.

PULMONARY TUBERCULOSIS.

TUMOUR PRESENT.—Diagnosis from: (1) *Gastric ulcer* with fibrosis and thickening, especially if pylorus. Final diagnosis rests with hæmatemesis. (2) *Gall bladder.*—In cancer, jaundice often early, but diagnosis may be difficult. Use X rays and opaque meal. (3) *Liver*, *spleen*, *mesenteric*, and *other tumours*. (4) *Pericholecystitis.*—Rare.

TUMOUR OF CARDIA.—Often palpable, or under left costal margin. Pain, vomiting, regurgitation rapidly following food. Wasting extreme. X rays and opaque meal reveal obstruction.

Treatment.—

SURGICAL.—Operation contraindicated only by secondary deposits, e.g., jaundice, or by very advanced state, not by palpable tumour.

OPERATIONS.—(1) Excision of growth, and gastro-jejunostomy. (2) Gastrojejunostomy alone, if tumour irremovable. (3) Gastrostomy (cancer of cardia). Results are improving.

Careful medical treatment *before and after operation.*

MEDICAL.—General treatment as in dyspepsia, modified to circumstances. Lavage if dilatation. Acids and ferments.

DIET.—Small, frequent feeds; especially peptonised milk, custards, etc. Avoid meat.

PAIN.—May need morphia, conveniently as tablets gr. ¼ under the tongue, one or two in twenty-four hours.

HÆMATEMESIS.—Treatment rarely of effect. Injection of morphia, ice to suck, rest.

OTHER FORMS OF GASTRIC TUMOUR.

SARCOMA.—Very rare. Usually under 25 years old. Rapid growth, large size, but without attacking mucous membrane, and hence no vomiting or hæmatemesis.

INNOCENT TUMOURS.—Polypi, adenomata, and various tumours are recorded. Mainly of pathological interest only.

HYDATID CYSTS.—Not very rare.

FOREIGN BODIES.—Hair tumour.

XII. CONGENITAL HYPERTROPHY OF THE PYLORUS.

(Congenital Hypertrophic Stenosis of the Pylorus.)

Etiology.—

AGE.—Onset of symptoms commonest in 2nd to 4th weeks of life. May date from birth.

Note. Congenital stenosis producing symptoms in adults has been described, but is doubtful, and certainly extremely rare.

SEX.—More frequent in males. About one third occur in first children.

Pathology.—Thickening of pylorus, due mainly to hyperplasia of muscular coat, especially circular layer. Stomach wall also thickened, especially near pylorus; may be some dilatation.

THEORIES.—(1) Congenital hypertrophy. (2) Early spasm of pylorus with secondary hypertrophy and stenosis. Thomson suggests failure of the normal relaxation of the pylorus due to incoordination, or infancy. Possibly pancreatic insufficiency affecting relaxation of sphincter (Eliakim). No constant hyperchlorhydria.

Spasm is undoubted factor, since (1) Cases recover while tumour is still palpable. (2) Obstruction varies from day to day.

Symptoms.—

VOMITING.—*Sudden, forcible, and often copious*; onset commonest in 2nd to 4th week; less often from birth; frequency variable.

WASTING.—Becomes extreme.

CONSTIPATION.

Pain and colic common.

Depending on above symptoms: *cyanosis, convulsions, subnormal temperature.*

Physical Signs.—Characteristic are:—

VISIBLE PERISTALSIS.—Especially after food; large waves passing left to right, often several waves visible. Repeated examinations may be necessary.

PALPABLE TUMOUR.—Firm, hard, movable tumour in position of pylorus; also best felt after food.

Dilatation is variable, usually absent.

Diagnosis.—Characteristics: (1) Onset under six weeks, (2) Chronic forcible vomiting with constipation and not diarrhœa, (3) Wasting; (4) *Visible peristalsis*; (5) Tumour. X rays with opaque meal will confirm.

Treatment.—

MEDICAL.—Often successful. (1) Lavage, morning and evening. (2) Feeding, ℥j per hour. Rectal saline, if necessary.

If loss of weight continues, operate. Recoveries occur even in very late stages, but *operation must not be long delayed* if wasting continues under treatment.

SURGICAL.—Rammstedt's operation: division of the muscular layer. Good results. Feeding after operation needs great care.

Alkalis to neutralize hyperacidity, e.g., sodium citrate (gr. ij) added to each feed.

In late stages mortality is high with either medical or surgical treatment; relative advantage in early stage still undecided. Various [illegible] differ vastly in their [illegible] on result of treatment. If recovery occurs, health in later life is good.

XIII. HÆMATEMESIS.

(Hæmorrhage from the Stomach.)

Etiology.—

LOCAL DISEASE OF STOMACH.—

1. GASTRIC ULCER.—Common. (Occasionally gastrostaxis (Hale White).)
2. CANCER.
3. DUODENAL ULCER.—Not common.

Occasionally.—

4. ACUTE GASTRITIS.—Streaks of blood only.
5. ABDOMINAL OPERATIONS.—Especially involving appendix or omentum; confined to severe forms and due to gastric erosions of septic origin.
6. Disease of blood-vessels, malar aneurysms, varicose veins.

PASSIVE CONGESTION OF THE PORTAL SYSTEM.—(1) *Cirrhosis of liver*—common (usually from veins at cardia). (2) Splenic anæmia. (3) Chronic disease of heart or lungs. (4) Tumours pressing upon, or thrombosis of, portal vein.

BLOOD SWALLOWED.—Origin: from nose, pharynx, lungs, or œsophagus.

Occasional causes.—

TRAUMA.

CORROSIVE POISONS AND GASTRO-INTESTINAL IRRITANTS.—Strong acids and alkalis, phosphorus, arsenic, antimony, etc.

TOXIC.—(1) Specific fevers: yellow fever, small-pox, malignant scarlet fever. (2) Various toxæmias, e.g., acute yellow atrophy.

DISEASES AFFECTING THE BLOOD.—Occasionally in severe anæmias, e.g., pernicious anæmia, malaria, leukæmia, and any

Hæmatemesis—Etiology, *continued*

splenic enlargement. (Splenic anæmia, from varicose veins.) Very unusual in hæmophilia.

RUPTURE OF ANEURYSM. Aorta or branches.

MALINGERING.

Has also been recorded in various nervous affections, e.g., hysteria, epilepsy, and occurs in new-born infants.

Profuse Hæmorrhage. Commonly due to (1) Gastric ulcer, or gastrostaxis; (2) Cirrhosis of liver.

Rare causes of profuse and fatal hæmorrhage: splenic anæmia, ruptured aneurysm, abdominal sepsis.

Rarely profuse or fatal in other forms.

Morbid Anatomy. In fatal cases always general anæmia.

STOMACH.—In gastric ulcer, cancer, corrosive poisoning, disease of vessels. In toxæmia cases, hæmorrhages into mucosa.

In obstruction to portal system: [illegible] pale, no lesion; œsophageal veins often not dilated. In gastrostaxis and miliary aneurysm, no bleeding point may be found, a careful examination may reveal minute erosions.

Symptoms.—Apart from vomiting of blood, are mainly due to the resultant anæmia.

CHARACTER OF VOMITED BLOOD. Usually dark, clotted, and may be acid, fluid or bitter. Altered by gastric juice depending on time in stomach [illegible] ground vomit.

AMOUNT. May be several pints.

ON OCCURRENCE OF HÆMATEMESIS INTO STOMACH. Fainting, common, cold sweat, collapse. [illegible] blood [illegible] rapidly [illegible] hæmorrhage [illegible] frequently [illegible] present [illegible].

Rarely convulsions in marked anæmia from [illegible].

Hæmatemesis may be first symptom in leukæmia, and rarely in gastric ulcer.

Diagnosis. Questions arising are:

(1) IS COLOUR DUE TO BLOOD? Discolouration usually occur from iron, bismuth, fruit juice. Microscopic and chemical tests.

(2) SOURCE OF THE BLOOD. Patient is usually a guide as to whether the blood is vomited or coughed up.

Hæmatemesis	Hæmoptysis
History suggestive of gastric or abdominal disease	Pulmonary or cardiac disease
Blood vomited	Blood coughed up
Airless, dark, acid, usually clotted	Frothy, red, fluid, alkaline
Sputum not stained after twenty-four hours	Sputum stained for several days
Melæna may occur	

SWALLOWED BLOOD. Origin often recognizable in nose, throat, or mouth.

XIV. NEUROSES OF THE STOMACH.

(*Nervous Dyspepsia.*)

Gastric disturbances without gross anatomical changes occurring in persons of nervous temperament or with definite neuroses, e.g., hysteria, neurasthenia. Rare before adult life, and commoner in women.

CLASSIFICATION.—(1) Motor; (2) Secretory; (3) Sensory. But mixed forms are usual.

Motor Neuroses.—

NERVOUS VOMITING.—Usually neurotic women. Food regurgitates without nausea or retching. Often in mouthfuls. Pain usually after meals, often immediately follows ingestion. Gastric analysis normal. Progress: (1) No wasting, suggestive of diagnosis; (2) Persistent and occasionally fatal, but recovery usual.

TREATMENT.—Food must be swallowed again. Urge patient to retain regurgitation. Usually there is much air swallowing.

NERVOUS FLATULENCE.—(1) Nervous eructations, aerophagia. May be explosive, and duration of several days. Is swallowed air. Hysterical women or occasionally children. Sometimes acquired by observation of others. May be painful distention of stomach, 'pneumatosis', if orifices do not relax. (2) Excessive peristalsis. Borborygmi, gurgling, and consciousness of peristalsis after meals often some extent atonicity. Intestines also often affected.

TREATMENT.—Weir Mitchell treatment if severe. Solution. Pressure on epigastrium. (See p. 'Diagnosis' or 'Dyspepsia', p. 386.)

Less important:—

HYPERMOTILITY.—Stomach empties rapidly. Shown by N. or stomach tube. May occur without symptoms. Often associated with other conditions, e.g., hyperacidity.

MERYCISM OR RUMINATION.—In advanced neuroses and idiots. No affection of health.

Secretory Neuroses.

HYPERCHLORHYDRIA.—Excessive percentage of HCl in gastric juice during digestion. Common form of dyspepsia in neurotic and also in chlorotic girls.

SYMPTOMS.—Onset follows meals after interval (½ to 2 hours), digestion active; epigastric pain, and eructations relieved by vomiting; constipation. Usually plump and appetite good, but 'afraid to eat'.

Is indefinitely distinguished from functional hyperchlorhydria.

Rare forms:—

GASTROSUCCORRHŒA.—Hypersecretion. Two forms: (1) Intermittent; (2) Continuous. Usually, but not invariably, hyperchlorhydria present.

INTERMITTENT HYPERSECRETION.—Rossbach's 'gastroxynsis'. Onset independent of meals, as at night; epigastric pain, and

Secretory Neuroses of the Stomach—Gastrosuccorrhœa, *continued.*

headache, followed by copious vomiting of acid fluid. Usually severe neurasthenia. Duration, few days. Resembles gastric crises.

CONTINUOUS HYPERSECRETION.—Reichmann's disease. More common. Constant vomiting, with pain and eructations, results in wasting. Dilatation from fluid and pyloric spasm. Condition suggests carcinoma.

TREATMENT.—As for hyperchlorhydria. Onset of intermittent form aborted by solid food. In continuous form, small frequent meals.

ACHYLIA GASTRICA NERVOSA.—Hypochlorhydria occurs not very rarely in nervous dyspepsia. Rarely complete absence of HCl and ferments, as in achylia gastrica, but subsequently returning. Symptoms usually severe (*see* ACHYLIA GASTRICA).

Sensory Neuroses.—

GASTRALGIA.—All forms of gastric discomfort occur with nervous temperaments, including severe paroxysmal pains. Hyperchlorhydria may co-exist.

TYPE OF PATIENT.—(1) Women at menopause, with worry and ill health. (2) With neurasthenia and hysteria. Occasionally at puberty.

SYMPTOMS.—In the paroxysmal form, severe epigastric pain radiating to back, onset sudden. No definite relation to food, may occur at night, vomiting uncommon. Food may either ease or aggravate. Pressure usually relieves.

DIAGNOSIS.—Needs great care. Severe paroxysmal pains also occur in (1) Organic disease of stomach, (2) Referred from other organs, e.g., gall-stone colic, epigastric angina, (3) Gastric crises. In neurosis (1) General signs of neurosis, (2) Vomiting infrequent, (3) Attacks intermittent.

BULIMIA.—Excessive hunger, often suddenly at night. Consumption of food either small or very large; in latter case dilatation occurs. In hysteria and neurosis. Similar attacks in hyperchlorhydria. Also diabetes.

AKORIA.—Constant hunger. Stomach never replete.

ANOREXIA NERVOSA.—Rare but fatal form. (*See* HYSTERIA.)

Treatment.—As a preliminary, look for (1) Physical and mental strain, e.g., worry, overwork. (2) General neurasthenic and hysterical symptoms. (3) Reflex irritation, e.g., eyes, pelvic and other diseases.

GENERAL PRINCIPLES.—Treat (1) General condition and constipation. (2) Prominent gastric symptom, e.g., pain, hyperacidity, alleviate as simply as possible (as in DYSPEPSIA). Careful consideration of relative importance of general condition and local symptoms. Serious cases with definite neurasthenia often best on *Weir-Mitchell treatment*, with diet of same, and not as suggested by gastric symptoms. Milk diet in general contraindicated, solids better. Lavage with caution: not to be done by patient.

XV. CYCLICAL VOMITING.

(*Periodic Vomiting*)

Recurrent attacks of vomiting occurring or commencing in childhood, usually accompanied by headache and evidences of acidosis.

General Description.

AGE AT ONSET.—Commonly 3 to 10 years.

ATTACKS RECURRENT.—Some periodicity, often 3 or 4 weeks, but intervals rarely regular and frequently longer.

ONSET.—Usually sudden without prodromata. Subject wakes with symptoms. May be irritability and heavy breath on previous day.

DURATION.—1 to 5 or 6 days.

VOMITING.—Forcible and repeated. Usually no nausea and no definite gastric pain. At first food, later bile.

HEADACHE.—Other symptoms may precede vomiting; usually frontal and bilateral. May be absent.

ACETONE.—Acetone and diacetic acid in urine; may be recognizable in breath.

DURING ATTACKS.—Constipation obstinate. Tongue coated. Breath heavy. May be diarrhœa. Temperature variable. Unable to take food and sometimes fluids. Becomes pale, drawn and prostrate.

BETWEEN ATTACKS.—Health often good and recovery rapid but if attacks are frequent patient becomes anæmic and pale and digestion is impaired.

Progress and Prognosis.—Attacks usually diminish or cease during puberty. May persist in less troublesome degree as migraine or more often as migraine. Attacks rare. Rarely fatal.

Pathogenesis.—Doubtful. Heredity common. More frequent in but not confined to highly strung, overstimulated, sedentary children. Appendicitis and coli bacillaria are put for occasional apparent case, but certainly not usual cause. No obvious connection with protein hypersensitiveness. Acidosis not regarded as initial factor. Probably hepatic deficiency connected with metabolism of fats and carbohydrates. Mild degrees common.

Treatment.—

DURING ATTACK.—Complete rest. Vomiting usually prevents drugs by mouth. Enema. Fomentation or mustard leaf to epigastrium. Ice to suck, or hot water. Food not to be pushed, but attempt small frequent drinks of fluids. Saline per rectum if collapsed.

BETWEEN ATTACKS.—No reliable method of prevention known. Sugar effective sometimes, e.g., two lumps of sugar after meals in water. Alkalis should be given, dosage not too large, e.g., sodium bicarbonate gr. x t.d.s. Constipation to be corrected.

CHAPTER LXXVI.

DISEASES OF THE INTESTINES.

I. CHARACTER OF STOOLS: NORMAL AND ABNORMAL.

Normal Fæces.—

WEIGHT.—About 5 to 6 ounces daily (140 to 180 grammes).

COLOUR.—Brown, due to stercobilin (identical with urobilin). Unaltered bile-pigment not present, being absorbed from intestine; stercobilin is altered bile-pigment excreted from gut wall.

ODOUR.—Fæcal, but only slightly offensive.

CONSISTENCY.—Firm and formed.

REACTION.—Faintly alkaline, or faintly acid, to litmus.

WATER.—Forms about 75 per cent.

PROTEIN.—None on ordinary diet.

FAT.—Forms 20 to 25 per cent. of dry solids. Also a proportion of neutral fats and fatty acids; sodium and potassium salts (soaps).

UNDIGESTED FOOD.—Present mostly in small quantity only. Vegetable débris and cellulose.

MICROSCOPICAL.—Unaltered food; epithelial cells; mucus. Crystals of calcium phosphate and oxalate, and in anomalies cholesterin and Charcot-Leyden crystals.

These characters apply to an adult on a mixed diet; on a diet rich in vegetable and starchy food the quantity is larger and the consistence softer, water forming 85 to 95 per cent. On a diet rich in animal food, the quantity is smaller and the consistence firmer, water forming 60 to 70 per cent.

A child of one year passes about 3 ounces daily. All of the amounts are subject to considerable individual variation.

Abnormalities of the Stools.—In constipation, scybala or hard masses. Ribbon-shaped stools occasionally in disease of sigmoid, and also from contraction of anal sphincter apart from disease of intestine.

Practically, abnormalities of stools are connected with diarrhœa, with few exceptions.

CONSISTENCE.—A loose motion in an adult is abnormal.

COLOUR.—

GREEN OR YELLOW GREEN.—Small and large gut both affected (from rapid peristalsis); especially in children, and after mercury.

YELLOW.—Senna, rhubarb, or santonin may cause such colour.

DARK OR BLACK—(1) Bismuth, iron (sulphides). (2) Blood, origin from above cæcum, but cannot be further localized.

CLAY COLOURED—(1) From absence of bile pigment in biliary obstruction; is accompanied by jaundice and bilinria, colour partly due to increase of fat (fatty acids). (2) From absence of pancreatic secretion causing failure to split neutral fats.

ODOUR. Offensive odour from putrefaction (bacterial decomposition mainly by anaerobes) of proteins, and production of amino bodies, indole, skatole, etc.

REACTION. *Acid* if much decomposition of carbohydrates (mainly children).

MUCUS. Large quantities have origin from colon or rectum, usually as long strips. In (1) colitis, acute, ulcerative, dysenteric, etc. (2) Mucous colitis. (3) Cancer of colon or rectum. Partly occur in disease of Fallopian tubes. In follicular ulceration of small intestine, usually in large masses.

BLOOD. (1) Red streaks from anus and rectum. (2) Mixed roughly and coloured, mucous, from colon. (3) Black stools, i.e., melæna* origin between stomach and cæcum.

PUS. (1) Ulcerative lesions of colon. (2) Diseases of rectum. (3) Carcinoma of large intestine. Large amounts usually from perforation of abscess, strangulated hernia, appendix, broad ligament.

MEMBRANES OR CASTS. Mucous. In (1) Membranous colitis. (2) Cancer of colon. (3) Fallopian tube, ovarian disease, etc.

FAT. Excessive fat in stools (steatorrhœa). Pancreatic secretion digests neutral fats, but bile secretion and fatty acids process and help in absorption of neutral fats; steatorrhœa depends on bile, see Constipation. Hence (1) Bile deficient, (2) Pancreatic secretion deficient, (3) Excessive fat, mainly *fatty acid*. (2) Bile normal and pancreatic secretion sufficient, but fat excessive, mainly *neutral fat*.

UNDIGESTED FOOD. Abnormal quantities in cases of excessive milk diet, or cases in which [illegible]

ABNORMAL SUBSTANCES. Material which has passed through stomach. Vegetable and food debris. Animal parasites. Gallstones. Mucous casts. Intestinal scales. Fragments of tissue, rarely in very rapid sloughing.

ABNORMAL SUBSTANCES (microscopic). Undigested muscle fibre (visible striations). Excessive undigested fat, refractile particles. Pus cells. Red blood cells. Ova. Protozoa and cysts.

Special Tests, etc.

Occult blood test and spectroscopic examination reveal small quantities of blood, and distinguish from iron or bismuth.

Excessive bacterial decomposition in small intestine is indicated by excess of ethereal sulphates and indican in urine.

Protein may be present.

* Melæna is properly plural.

Character of Stools in Special Conditions.—

TYPHOID FEVER.—'Pea-soup' stool; loose, uniform, light brown.
CHOLERA.—'Rice-water' stool: very watery, practically no smell or fæcal matter.
DYSENTERY.—Mucus and blood (*see* p. 90).
OBSTRUCTIVE JAUNDICE.—Clay-coloured stool.
HÆMOLYTIC JAUNDICE.—Stool of normal colour.
MUCO-MEMBRANOUS COLITIS.—Mucus, and 'membranes' or 'casts'. Occasionally intestinal sand.
PANCREATIC INSUFFICIENCY.—Light colour, frothy, bulky, [illegible] greasy stool. Also in coeliac disease (q.v.).

[illegible]FIGHT.—**Inflammations and Character of Stools.**—

[illegible]OLOUR.—Brown. OF SMALL INTESTINE.—Colour greenish yellow. Unaltered bile, mucus slight, undigested food. Diarrhœa not necessarily; stercobilin is altered; water absorption in colon may solidify stool.
[illegible]OUR.—Fæcal. OF LARGE INTESTINE.—Gray or brown, thin, [illegible]NSISTENCY.—Mucus excessive.
[illegible]ACTION.—Nothing distinctive.
[illegible]ATE.—JEJUNITIS.—As in small intestine.
[illegible]TROP[illegible]
[illegible]LAT.—
[illegible]

II. TYMPANITES.

Gaseous distention of the intestines occurs in: (1) Dyspepsia, gastritis, common, transient. (2) Acute abdominal disease, obstruction, and after operations. (3) Acute infective fevers: pneumonia, etc. The retention of the gases of putrefaction (q.v.) may be due to: (i) Paresis of intestinal muscle. (ii) Obstruction. (iii) Diminished absorption. (iv) Increased formation.

Symptoms and Signs.—Abdominal distention, often painful. Tympanic resonance. Diminished respiratory movements. Diaphragm pushed up. May be passage of flatus. Visceral sequelæ: Heart displaced upwards, beats faster, rapidly fatal cardiac failure, especially if diseased. Also respiratory embarrassment.

Treatment.—

I. IN ABSENCE OF ACUTE ABDOMINAL CONDITIONS.—
i. LOCAL TO ABDOMEN: Hot fomentations. Turpentine stupes. Gentle massage.
ii. BY MOUTH (of little use in serious cases).—Essential oils, e.g., of cajeput ♏j to ♏ij on sugar; turpentine ♏v to ♏x, hourly; sal volatile ♏x, half-hourly, 6 doses.
iii. ENEMA.—Turpentine ʒss to ʒij. Prepare a soap and water enema, and divide into two parts: to one half add the oil and inject, and follow shortly with remaining half.
iv. PITUITARY EXTRACT, e.g., PITUITRIN.—Hypodermic injection of 1 c.c. Most powerful and effective treatment. Two or three injections at intervals of 4 hours.

2. ACUTE ABDOMINAL CONDITIONS.—Operation.
The passage of a long rectal tube is useless.

III. DIARRHŒA. ENTERITIS. COLITIS.

(*See also* DIARRHŒA IN CHILDREN p. 426)

Diarrhœa is commonly, not invariably, associated with [illegible] catarrh of varying extent.

CAUSES OF DIARRHŒA.

(1) Primary. (2) Secondary. (3) Special types.

Primary Diarrhœa.

DIETETIC. [illegible] water. Idiosyncrasy [illegible]

CONSTIPATION. [illegible]

TOXIC SUBSTANCES. [illegible] mercury [illegible]

CHANGES OF WEATHER OR OF CLIMATE. [illegible] mode of [illegible] Chill [illegible] Heat [illegible] hance of [illegible] excessive warmth.

ALTERATION OF INTESTINAL SECRETIONS. [illegible] pain [illegible] little known [illegible] injunctive [illegible]

NERVOUS DIARRHŒA. [illegible] compare [illegible] or secretion [illegible]

BACTERIAL INFECTION AND INTOXICATION. In severest forms [illegible] quently [illegible] certainly bacterial [illegible] incompletely [illegible] milder type.

Secondary Diarrhœa.

INFECTIVE CONDITIONS. (a) [illegible] enteric, and numerous tropical [illegible] sprue, hill diarrhœa. (b) [illegible] pneumonia (severe symptom [illegible]).

Occasionally tapeworms.

CHRONIC CIRCULATORY DISTURBANCES. [illegible] congestion, cirrhosis of liver, chronic heart and lung disease, often obstinate diarrhœa.

DISEASES OF INTESTINES OR NEIGHBOURING PARTS.—Cancer, chronic peritonitis, often alternating with constipation.

LARDACEOUS DISEASE.

TERMINAL CONDITIONS AND CACHEXIA.—Cancer, nephritis, severe anæmia, etc.

Diarrhœa, *continued.*

RELATION OF APPENDICITIS TO ACUTE AND CHRONIC COLITIS, etc.—Above symptoms, in severity rarely exceeding medium, and usually mild, frequently associated with symptoms of appendicitis, probably from simultaneous involvement. Such colitis frequently persists or relapses, and often so even after appendicectomy. *Procedure recommended*: an interim appendicectomy with warning to patient of temporary persistence of symptoms and tendency to relapse of colitis; the operation should be regarded as a preliminary to medical treatment of the colitis; appendix often shows little change.

RELATION OF IDIOPATHIC ACUTE COLITIS TO ULCERATIVE COLITIS.—(1) Symptoms similar. (2) Morbid anatomy: in early ulcerative colitis resembles acute colitis, viz., reddened inflamed mucous membrane.

(Rapid ulceration is very uncommon under 20 years of age apart from bacillary dysentery.)

CONCLUSIONS.—(1) Both conditions almost certainly bacterial. (2) Possibly one or more kinds of different bacilli specially associated with ulceration; at present no specific bacilli identified (dysentery not here referred to). (3) No essential difference in symptoms or origin; a big one in treatment.

RELATION OF ULCERATIVE COLITIS TO DYSENTERY AND ACUTE COLITIS.—Symptoms and morbid anatomy: in bacillary dysentery and ulcerative colitis identical. Where dysentery is present, differentiation depends upon bacteriological examination. Similarly for acute colitis.

ASYLUM DYSENTERY.—Outbreaks are frequent in asylums. Symptoms, morbid anatomy, etc., identical with ulcerative colitis. No specific organism proved; but recent and more complete investigations, as by Dudgeon and Sherrington, have indicated bacilli of the Flexner group.

FIREMAN'S CRAMP.—Occurs in stokers and others who spend periods in highly heated atmosphere and drink much cold water: frequent watery stools, with marked collapse and severe muscular cramps. Considerable resemblance to cholera.

DIAGNOSIS OF AFFECTED SITE.

Duodenum.—Symptoms indefinite. Jaundice and gastritis.

Small Intestine.—

PAINS.—Colicky; diffuse tenderness.

DIARRHŒA.—Stools not necessarily very frequent, and may be no diarrhœa. Flatus often marked.

STOOLS.—Yellow to brown or green; undigested food; little mucus or blood.

Large Intestine.—

PAINS.—Especially with motions (*tenesmus*).

DIARRHŒA.—Motions extremely frequent.

STOOLS.—Blood and mucus.

COLON.—Tender.

TREATMENT.

Acute Diarrhœa: Acute Enteritis.—

GENERAL HYGIENE. Bed, if pyrexia or weakness, until temperature normal, and stools formed: *keep warm*, including limbs.

DIET. If severe: Milk and lime water, or whey or albumen water with fluid *ad lib.* (in small drinks). If milder: Semi-fluids (custards, etc.). Avoid solids and hot food.

DRUGS. Indications are: (1) Remove irritant. (2) Reduce irritation and inflammation present.

(1) *Initial dose* of castor oil (ʒj.): if much pain add tinct. opii ♏xx. Or repeated smaller doses (ʒjs. or pulv. rhei co.). Castor oil specially indicated when origin from food or dyspepsia, but contraindicated by excessive purging.

(2) To arrest diarrhœa and peristalsis: not advisable under forty-eight hours unless severe.

(a) Chalk Mixture (Mist. cretæ B.P.) ℥ss., two to four hourly: tinct. opii ♏xx may be added or pulv. cretæ aromaticus c. opio gr. xx. four hourly.

(b) Bismuth and Opium.

℞ Bismuthi subnitrat. [illegible] ʒj
Tinct. Opii [illegible]
Mucilag. [illegible] ℥ss
[illegible]

Other drugs: [illegible] ♏x [illegible] plumbi [illegible] pulv. kino [illegible] pulv. catechu [illegible]

(c) Enema: Starch mucilage ℥ss. and opium tinct. opii ♏xx. [illegible] and [illegible]

ALCOHOL: Brandy only [illegible] often effective [illegible]

SPECIAL TREATMENT:

1. PAIN: Warmth to abdomen: opium by mouth. With severe colic or tenesmus inject morphia gr. ¼.
2. VOMITING: Ice to suck. Iced peptonized milk.
3. FLATULENCE AND DISTENSION: Carminatives (see Dyspepsia). Turpentine enema (see [illegible]). Intestin.
4. STIMULANTS: Red champagne. Brandy if exhausted, often causes nausea.
5. COLLAPSE: Stimulants alcohol (ether injections), camphor, or caffein sodio salicylate, strychnine, etc. Inject hypertonic saline solution (1·8 per cent.) subcutaneously or intravenously.

PROGRESS.—As diarrhœa and pain diminish, increase diet by eggs, milk, fools, broth, jelly. May commence with peptonized milk. Avoid gruel, arrowroot, hot fools. For persistence of diarrhœa, see CHRONIC DIARRHŒA.

Acute Colitis.—Treatment mainly as above, but drugs by mouth of less and enemata of greater value.

ENEMATA.—(1) Starch and opium enema. (2) Rectal washes:

Diarrhœa—Acute Colitis Treatment, *continued*

sod chlor or sod bicarb (ʒj to Oj) at 98°. Silver nitrate and similar injections when improving (but not in acute stage)

Chronic Diarrhœa. - Investigate as preliminary, so far as possible (1) Etiological factor (2) Locality affected by noting (*a*) Symptoms (*b*) Excreta in fæces mucus, blood pus parasites undigested food, in urine sulphates indican (*c*) Rectal examination, sigmoidoscopy. (4) Associated diseases appendicitis neoplasms, visceroptosis adhesions

GENERAL TREATMENT - Rest in bed with low diet of itself often cures or relieves but great tendency to relapse Period in bed valuable in all cases at onset of treatment

If bed impracticable, and when getting up *feet and general hygiene* framed as in chronic dyspepsia A simple mixed diet is preferable milk is often tolerated badly Avoid chill

SPAS Plombiers Harrogate etc routine and douche treatment valuable in chronic enteritis and especially colitis

Chronic Diarrhœa with Dyspepsia. Treat as dyspepsia If necessary add drugs as in enteritis

Chronic Enteritis. Especially in absorbing [illegible]

MILD — Saline aperient in morning

SEVERER FORM

ASTRINGENTS Follow [illegible] ℞ [illegible] Mix [illegible] ʒss tabs, [illegible] gr xx, tabs ℞ [illegible] ℞ Tinct chloroformi et morphinæ [illegible] ℞ [illegible] ℞ Tannin [illegible] Tannigen [illegible] Bismuth sulphanilate and

Prescription [illegible]

℞ Acid Sulphuric Aromat [illegible] ♏ x
Tinct Chloroformi et Morphinæ [illegible] ʒ
Tinct [illegible] ♏ xx
Aq Chloroformi [illegible] ad ℥ x

Other common and valuable astringents Tabs [illegible] gr xx to xl pulv kino [illegible] opium [illegible] pul plumbi c opio gr [illegible] to xx [illegible] ♏ xx to xl [illegible] ʒj not with [illegible]

INTESTINAL ANTISEPTICS Less valuable [illegible] effective In contrast use bismuth salicylate gr x to xx salol in cachets, gr v to x

Chronic Colitis (Catarrhal).

DIET.—Avoid meat but order liberal diet to give little residue

DRUGS By mouth of little use Astringents may be employed as for 'enteritis'

ENEMATA AND LAVAGE.

℞ Starch and opium daily, for few days

℞ Bland rectal washes sod chlor or sod bicarb ʒj to Oj, 3 pints at 98°, with long tube

(3) Medicated injections: empty colon by rectal wash two hours previously. If painful, inject morphia (hypodermically). Silver nitrate (gr. ss in ℥j) 2 to 4 pints, albargin (gr. j in ℥j), 1 to 1½ pints daily, frequently effective.

SPA TREATMENT. *See* CHRONIC DIARRHŒA, p. 422.

APPENDICOSTOMY. In obstinate and failing cases. Colon washed through daily for months. Results fair, but not advisable before prolonged medical treatment.

Nervous or Hysterical Diarrhœa. Change of surroundings. Instruct to resist the inclination to movements of bowels. Bromides. Treat psychical condition. Weir Mitchell rest cure, if severe.

Pancreatic Diarrhœa. Reduce fats in food. Give diastase and pancreatic ferment.

Lienteric Diarrhœa. Use arsenicals. Hypodermically with meals, orally half hour before meals.

Morning Diarrhœa. Avoid exposure and chill overnight; no fluids after 7 to 8 p.m.

Suppository in Diarrhœa. [illegible]

℞ Extract Belladonna gr. [illegible]
Morphine gr. [illegible] Cocoa Butter [illegible]

Constipation. Frequently follows or alternates with diarrhœa. Enemata. ℥j to ℥iv. Infusion of senna pods. Simple enemata. Avoid purgatives and laxatives.

Notes on Certain Drugs in Diarrhœa.

* OPIUM. Is most powerful drug. [illegible] to health. [illegible]

ACTION OF CHALK. [illegible] tissues, [illegible] flow, contract. [illegible] of mucous membranes and [illegible] ✔ ✔

IV. ULCERATION OF THE INTESTINE.

Occurrence. Ulcers of the intestine existing peptic ulcer in —

SPECIFIC INFECTIONS. Typhoid, dysentery, tuberculosis, syphilis.

ULCERATIVE COLITIS.

STERCORAL ULCERS. With stercoral constipation from impaction of scybala. Diverticulitis may follow.

FOLLICULAR ULCERATION. In children and in secondary and terminal diarrhœa. Small ulcers with sharp edges, never perforate. From swelling and degeneration of follicles. Never diagnosed.

Rare forms —

FOREIGN BODIES, NEOPLASMS AND EXTRAFOUS ABSCESS.

SIMPLE PERFORATING ULCER.—Solitary ulcer may perforate jejunum, cæcum, or colon. Very rare.

Ulceration of the Intestine, *continued.*

Symptoms.—Intestinal ulceration is suggested by: (1) Diarrhœa; (2) Abdominal pain, either (a) colicky, or (b) colonic tenderness and tenesmus if colon affected; (3) Hemorrhage from rectum; (4) Stool may also show (a) mucus, (b) pus, (c) fragments of tissue.

Perforation may give rise to general peritonitis or local peritoneal abscess.

V. ULCERATIVE COLITIS.

Symptoms, morbid anatomy, treatment etc. as in bacillary dysentery (see also Acute Colitis). Almost certainly a bacterial infection.

Etiology. Age 20 to 30 years. Sexes equal.

PREDISPOSING FACTORS. Usually none. Occasionally diarrhœa or constipation. Chronic interstitial nephritis in some.

Morbid Anatomy. Colon dilated, not hypertrophied. Ulceration predominates, often extensive, irregular, edges infiltrated, polypoid in nature, remaining mucosa moderately thickened and mucous membrane cherry red. In early and very acute cases *Rarely* fatty red and inflamed, ulceration slight.

Summary of Other abscesses. Multiple pyæmic abscesses with [illegible]

Symptoms. (1) *Diarrhœa*: stools very frequent, distended with mucus. (2) *Abdominal pain and tenesmus*: usually [illegible]. (3) Colonic tenderness. (4) Tongue clean or slightly coated; temperature variable, high or low. (5) Loss of weight in Recurrences [illegible] to the severity of symptoms and degree of collapse.

Progress. Remission or [illegible] to be expected.

1. [illegible]
2. [illegible] high or slights within three days in severe cases. Becomes chronic. Death frequent in three to nine months.
3. Tendency to relapse with intervening constipation.
4. Recovery [illegible].

Wasting. Debility. Anæmia. Bowel often total lesion.

VI. MUCO-MEMBRANOUS COLITIS: MUCOUS COLITIS.

Chronic condition of colon characterized by (1) Neurasthenia and neuroses, (2) Passage of mucous casts resembling membranes, (3) Constipation.

Etiology.—

AGE OF ONSET [illegible] years. *Duration* often many years.
SEX.—Five female to one male.
CAUSATION.—Unknown. *May be no inflammation of colon.* Productions of casts may be explained by excessive secretion of mucus, together with constipation, the resulting delay in

evacuation permitting time for coagulation of mucin by mucinase of intestinal mucous membrane (cf. BRONCHIAL ASTHMA).

CASE OF PATIENT. Thin anæmic woman, muddy complexion, poor appetite, some visceroptosis. Neurasthenia of some neurosis very common.

Symptoms. Patient's life consists of:

1. Periods of freedom (sometimes months). Health good.
2. Periods in which attacks occur. Few days to several months. Onset often follows worry or error of diet.

SYMPTOMS DURING ATTACKS:

1. CONSTIPATION. Obstinate. May be attacks of diarrhœa.
2. PAROXYSMAL ATTACKS of *Colic*, passage of stool and mucus (or *Pain or Membrane* usually occurs after several paroxysms) or *Tenesmus*, with membranes, severe.

   (a) [illegible] or formed motion with [illegible]. Mucus may be [illegible]. In masses or strips. (b) Membranes.

   Membranes: (1) Are flakes from strips to tubular casts [illegible]. (2) [illegible] appearance of smooth outside, rough [illegible] is [illegible] by pattern of skin. (3) May [illegible] epithelial.

   Intestinal "colds": common. [illegible] several days.

Complications and Associated Diseases.

NEURASTHENIA: [illegible] female [illegible] common. Mental [illegible].

HÆMORRHOIDS: [illegible] and more [illegible].

Diagnosis. [illegible] Mucus [illegible] and also membranes [illegible] cast [illegible] *rectum* [illegible] associated diseases.

Prognosis. Rarely fatal. Recurrences [illegible] to recur many years [illegible].

Treatment. [illegible] simply. Bed, one to two weeks at beginning of treatment of severe.

NEURASTHENIA: [illegible] may alone result in cure.

VISCEROPTOSIS: If present, abdominal belt and exercises, not massage.

TREATMENT OF COLON: [illegible] essential. Warm alkaline soda bicarb. wash [illegible] with long tube in morning, olive oil injection at night [illegible] to be retained overnight. Astringents and antiseptics by mouth useless, and purgatives harmful.

DIET: Simple ordinary diet is best. At Bonnières, simple diet is given. Some recommend coarse foods.

SPAS: Bonnières, Harrogate. Standard treatments valuable.

BILIOUS ATTACKS: Regular life and exercise. Aperients to be avoided if possible, especially saline. Safe and best are paraffin liq. ℥ss to ℥iss daily, infusion of senna pods, castor oil gss to j.

Diarrhœa, *continued*

VII. DIARRHŒA IN CHILDREN.

Etiology.—

AGE.—Commonest 6 to 18 months, especially about dentition.

SEASON.—Most frequent in summer, maximum in July.

DIET.—Important factors are: (1) *Bottle fed infants* very susceptible; (2) *Overfeeding*, acts by direct irritation, and also by increasing fermentation. (3) Excess of fat. (4) Presence of bacteria. In older children, special articles, e.g. over ripe or unripe fruit.

ENVIRONMENT.—Dirt, squalor, and lack of fresh air.

EPIDEMICS.—Common in institutions. Infections bacteria spread by food.

OTHER FACTORS.—(1) Chills. (2) Teething. (3) Rickets.

Bacteriology.—Various bacilli occur in epidemics and also in sporadic cases in summer, especially in artificial feeding. In England, the most frequent is Morgan's Nov. bacillus. In America various dysentery strains are recorded. The bacteriology, including Morgan's bacillus, is still uncertain. Milk from cows with mastitis has caused streptococcal outbreaks.

Morbid Anatomy.—*In intestines*.—Changes often slight even in severe forms.

SIMPLE DIARRHŒA.—Congestion and slight thickening of the mucous membrane.

ACUTE GASTROENTERITIS.—Mucous membrane thin and atrophied, solitary follicles less prominent, often no other marked change. Occasionally ulceration, redness, swelling.

In other organs may be: Fatty liver, etc. Blood appearances.

CLINICAL GROUPS.

(1) Simple non-inflammatory diarrhœa. (2) Acute gastro-enteritis or enterocolitis, inflammatory diarrhœa. (3) Cholera infantum, infantile cholera. The last two groups are varieties in severity of 'summer diarrhœa' or 'diarrhœa and vomiting' of children. (4) Chronic diarrhœa.

1. **Simple Diarrhœa.**—Usually from errors of diet, or from chill.

ONSET.—Often preliminary symptoms. Restlessness. Abdominal colic, legs drawn up, and abdomen hard.

PYREXIA.—Slight.

VOMITING AND DIARRHŒA.—Motions 2 to 4 daily. Stools offensive or sour, undigested food present, mucus in later stages, colour light brown to green.

PROSTRATION.—Usually moderate, but severe in feeble children.

DURATION.—Usually few days. May pass into severer types in summer. Tendency to recurrence or to gastric disturbance subsequently.

Notes.—*Green colour of stools* is variously ascribed to presence of bile pigment, from rapidity of peristalsis, to conversion of stercobilin into biliverdin by bacilli, and to chromogenic bacilli.

Lienteric Diarrhœa.—Motion follows taking of food. Usually in older children, 5 to 6 years, and subacute. Much undigested food in stools. Wasting occurs, and results may be severe from lack of nutrition.

Acute Gastro-enteritis. *Summer diarrhœa.*

ONSET.—Sudden, may be convulsion or fainting.
VOMITING.—Rarely absent, often persistent.
DIARRHŒA.—Stools numerous. At onset faecal, then watery, mucus usual but blood uncommon. Prolapse of rectum frequent. Intense excoriation round anus.
ABDOMINAL PAIN AND TENESMUS.—Legs drawn up, and abdomen hard, but later with collapse often becomes lax.
TEMPERATURE.—Usually 103° to 105°. Hyperpyrexia not infrequent.
WASTING, EXHAUSTION AND COLLAPSE.—Rapid and very severe. Eyes sunken, hollow, fontanelles depressed. Shrunken appearance. Dry skin. Cold and cyanotic though rectal temperature high. Stools fæcal and watery. Passes from early restlessness to condition of stupor, collapse with feeble incessant cry. Vomiting and even diarrhœa may cease.
URINE.—Extreme rare scanty. Stomatitis frequent.
MORTALITY.—Illy. Death from exhaustion less often than from pneumonia or hyperpyrexia. Very rarely meningeal rash.
CONVALESCENCE.—Very slow. Tendency to relapse and to chronic diarrhœa.

Choleraic Diarrhœa. *Infantile cholera.*—Exaggeration of type of above. Collapse takes place with great rapidity. Mortality very high.

Chronic Diarrhœa.—May follow any form. Loss of weight often progressive. Vomiting common. Death from exhaustion, or from tubercle or bronchopneumonia.

TREATMENT.

Prophylaxis.—Of great importance. (1) Avoid weaning in hot months. (2) Protect against chills. (3) Utmost cleanliness of bottles and teats. (4) Pasteurization of milk. (5) Amount of fat and strength of milk reduced on any intestinal disturbance. (6) Fresh air and attention to general health.

General Outline of Treatment.

HYGIENE.—Avoid chills, but good ventilation of rooms. Warm clothing, especially of extremities. Flannel abdominal binder. Sponging instead of bath.
DIET.—Only albumen water for twenty-four to forty-eight hours. Then whey, or white-wine whey. As the condition improves, Mellin's food may be added to whey, or meat juice given. Milk with lime-water or barley-water to be commenced with caution.
FLUIDS.—Freely. In addition to above, boiled water in small amounts (3j) frequently, every 15 minutes, as indicated.

Diarrhœa in Children Treatment, *continued.*

DRUGS —

1. *Initial dose* of castor oil if seen *early in attack* single dose up to ʒj, or small doses repeated in mild cases, viz.,

℞ Ol. Ricini ℥v Aq. Carui ad ʒj
Mucil. Acaciæ ℥x

Four-hourly. (Aids removal from intestine of irritating contents.)

2. Bismuth, chalk, and astringents (tinct. catechu) in various forms and combinations are the most efficient drugs. For child of 1 year —

℞ Bism. Oxycarb. gr. xxx to xl. Glycerini ℥ss to x
Pulv. Cret. Ar. mat.
c. Opio gr. i to ii Aq. Anethi ad ʒj
Tinct. Catechu ℥xxx to x
Every 4 hours.

Of 'intestinal antiseptics', calomel only is of accepted value. Best when combined with Dover's powder.

℞ Calomel gr. ¼ Pulv. Ipecac. Co. gr. ¼
Repeated 4 hourly, i.e. ipecac. four; calomel gr. j in 24 hours.

Note. A child should not be awakened for a dose containing opium.

AS CONDITION IMPROVES. Gradual increase of diet, avoiding cream, and sparing milk. With undigested food in stool, repeat castor oil or calomel.

'Lienteric Diarrhœa'. Due to amœbic peritonitis. Treat with arsenic and strychnine. Results good. For child of 1 year.

℞ Liq. Arsenicalis ℥ss Glycerini ℥x
Tinct. Nux Vom. ℥j Aq. Anethi ad ʒj

Small dose of opium may be added. Rest to peritoneum.

Severe Diarrhœa.—

INELASTICITY OF SKIN from withdrawal of fluids and high pyrexia are signs of severity. Indication is to combat the collapse and loss of fluid.

FLUIDS. Inject sterile saline solutions subcutaneously. Fluid bag or Spittal's thermos flask both suitable. Solution sodium chloride 50 or 100 grains hypertonic to plasma; calcium chloride gr. iv may be added. Inject 4 to 10 ounces repeatedly.

Intravenous injections are very rapidly absorbed, and with the lax skin are easily performed (H. Miller).

STIMULANTS. Brandy ʒss to ʒj in twenty-four hours, or champagne if much vomiting. Inject strychnine (gr. 1/30) four- to six-hourly. For acute collapse, mustard baths.

DIET.—Only boiled water for twenty-four hours, given cold, very frequently. Then albumen water. Acute stage rarely lasts more than two to four days, and children stand starvation well, nor in this condition can they digest food. Progress with food to be very gradual; commence with whey, broth, Mellin's food with fresh barley-water; special care in commencing milk. All food

given cold, in increasing amounts guided by improvement especially if vomiting

VOMITING. Wash out stomach, with catheter and funnel

DRUGS. Drugs as in simple form above but initial castor oil omitted. When mucus present in stool, give starch and opium, enemata (starch emulsion ℥ss tinct. opii ♏xl) twice daily, also for prolapse of rectum

HYPERPYREXIA. Irrigation of colon with cold water. First injection about 80 to 85° (4 ounces). If no collapse follows later injections can be given more cold. After injection wrap up well to prevent chill.

Chronic Diarrhœa. A course of treatment carried out as for acute diarrhœa; diet of albumin water and Mellin's food, raw eggs, bismuth and pulvis ipecac. et morphinæ, with or without opium; and utmost attention to prevent any chill. In resistant cases silver nitrate often valuable.

℞ Albumin water ... Tinct. Opii ... ♏xl; Argenti nitrat. ... gr. ss

In older children restriction of diet to be less severe; sugar and starch mainly to be avoided.

Occasionally a child is been overstarved and improves on increasing the diet; but fats and starches may be more

VIII. APPENDICITIS.

Etiology.

FREQUENCY OF APPENDICITIS PROBABLY DUE TO:

(1) Long narrow lumen; contents hard, dry, and easily blocked by fæces or foreign bodies; and mucous membrane injured.

(2) Single supplying artery; branches of such artery blocked; supply thus easily affected or insufficient to deal with inflammation.

AGE. Nearly 50 per cent before 20 years of age. Rare before 5 years.

SEX. Commoner in males.

FÆCAL CONCRETIONS AND FOREIGN BODIES. Soft fæcal moulds or hard concretions are both common; shape often resembling date stones or melon seed. Intestinal parasites, pins, and a great variety of small foreign bodies recorded.

ORIGIN OF INFLAMMATION. May be: (1) Foreign body, fæcal concretion, etc.; (2) Local inflammation, no exciting cause found; (3) Colitis or gastro-intestinal disturbance; (4) General infection present, e.g., tonsillitis, pneumonia, influenza. All groups common except the last.

POSITIONS OF APPENDIX.—In order of frequency: (1) To left of cæcum, in iliac fossa; (2) Hanging over brim of pelvis: important position from reference to pelvis of symptoms and signs; (3) In retrocæcal and ileocæcal fossæ: especially common in disease; (4) To right of cæcum.

Appendicitis, *continued*

Morbid Anatomy.—Appendices at operation may be classified, after Warren, as follows:—

1. CATARRHAL.—Peritoneal surface, little change; internally mucosa œdematous, with petechiæ or ulceration, especially distal half; fæcal concretions, etc., common.
2. PHLEGMONOUS.—Appendix red and swollen; lymph or pus on peritoneal surface; internally œdematous, no ulceration. Is a cellulitis, often streptococcal.
3. EMPYEMA.—Appendix dilated with mucopus; constricting point is usually stenosis from previous ulceration. Often lymph on peritoneal surface.
4. ULCERATIVE. Advanced ulceration at point of impacted concretion, empyema distally; may be perforated.
5. GANGRENOUS.—Appendix gangrenous, containing fæcal pus. Is advanced stage of first three varieties, or from kinking of blood vessels.

OBLITERATIVE APPENDICITIS.—Widespread ulceration may heal, and result in obliteration of lumen; a natural cure. Entire appendix sloughs off in rare instances.

TUBERCULOSIS OF APPENDIX. See TUBERCULOSIS OF THE INTESTINES.

Symptoms of Acute Appendicitis. (1) Sudden onset of abdominal pain settling in right iliac fossa; (2) fever and rigors, pulse; (3) Nausea, vomiting, and constipation; (4) tenderness in right iliac fossa.

PAIN.—Sudden abdominal pain (a) Commencing in right iliac fossa, or (b) At onset more diffuse, often central, settling in right iliac fossa. Usually sharp and severe; rarely dull ache. Rarely in left iliac fossa.

FEVER.—Some degree extremely constant. Moderate, rarely exceeding 102°. May be absent in (1) Localized abscess already formed; (2) severe general peritonitis; other signs definite. Rigors not common at onset.

PULSE.—Increase roughly proportional to temperature. Most valuable measure of progress: *increasing rapidity is a serious sign.*

GASTRO-INTESTINAL DISTURBANCES.

TONGUE.—Furred and moist; rarely dry.

VOMITING.—May be absent. Rarely after second day of full attack; persistence points to serious lesion.

CONSTIPATION. Usual. Diarrhœa occasionally in children.

Abdominal Signs.—

INSPECTION.—No alteration in earliest stages. Lack of movement develops on right side.

PALPATION.—(1) Deep tenderness at McBurney's point, most definite sign. (2) Increased resistance or definite rigidity of right rectus. (3) Often, an ill-defined swelling in right iliac fossa, occasionally a definite tumour from a pre formed abscess.

McBurney's Point.—Situated on line from umbilicus to anterior superior spine of ilium at outer edge of rectus. Corresponds to base of appendix.

SUPERFICIAL TENDERNESS.—In right iliac fossa or area of 11th dorsal segment. Due to, and only present with distention of appendix, and disappears on perforation.

VARIOUS PHENOMENA.—Right leg is often [illegible]. Irritability of bladder may be early symptom. With appendix hanging over brim symptoms suggest pelvic disease.

RECTAL EXAMINATION.—Never omit when in doubt or before deciding against operation. Usually nothing felt in early stages. But occasionally in pelvic position of appendix abdominal signs may be slight with tender mass palpable by rectum and rectal wall edematous on right.

Leucocytosis.—In mild cases [illegible] or none. In acute cases [illegible] constant. [illegible] with increased percentage of polymorphonuclears. [illegible] See Leucocytosis and Leucopenia.

Progress and Result of Attack.—May be (1) Recovery. (2) Appendix abscess formation. (3) General peritonitis.

MILD CATARRHAL ATTACKS.—Recovery in [illegible]. Symptoms subside in [illegible] to three days, and patient may walk. Appendix may return [illegible]. Recurrence frequent.

CATARRHAL FORM.—May pass [illegible] second time, e.g. [illegible] catarrh or phlegmonous [illegible]. Recovery with tendency subsequent [illegible]. Localized abscess or perforation. General peritonitis.

ULCERATION EXTENSIVE.—Abscess [illegible].

GANGRENOUS FORM.—Results in abscess or general peritonitis.

APPENDIX ABSCESS.—Process of inflammation and formation of pus may be limited by interperitoneal adhesions, and appendix abscess thus resulting.

DIAGNOSIS OF ABSCESS FORMATION DURING ATTACK.—(1) Increase in resistance and tenderness right iliac fossa (or per rectum). (2) Constitutional symptoms more marked, especially raped pulse and leucocytosis. Temperature usually rises but may be moderate, may be sweats.

SITES OF ABSCESS.—(1) In iliac fossa, roof formed by abdominal wall. (2) In pelvis, palpable through rectum or vagina. (3) Retrocolic. (4) In right flank.

TERMINATION OF ABSCESS (without operation).—(1) Ruptures into general peritoneal cavity, whence general peritonitis. (2) Discharges through rectum, intestine, or rarely vagina. (3) Tracks in various directions, e.g. subphrenic abscess. *Death* may result from general peritonitis, pylephlebitis, septicæmia, perforation of blood vessels.

GENERAL PERITONITIS.—May result from (a) Acute perforation of appendix and spread of inflammation early in attack, with or without unsuccessful attempts at localization. (b) Rupture of appendix abscess.

Appendicitis – Progress and Result of Attack, *continued.*

ACUTE PERFORATION.—Onset as in acute appendicitis, but becoming more severe, especially general symptoms. Onset sudden pain and tenderness more diffuse and localizing less; abdomen becomes distended and immobile; bowels not open, or possibly one motion. *Pulse progressively more rapid*; vomiting, dry tongue, Hippocratic facies, and development of usual signs of general peritonitis. Temperature rises, but often falls later. No localizing signs may occur.

RUPTURE OF ABSCESS.—Acute attack previously few days to three weeks; or indefinite gastric symptoms. Rupture followed by shock, collapse, sudden pain and development of general peritonitis.

It is essential to note that at and from the onset peritonitis may be present and be extending without any symptoms differentiating it from a mild catarrhal attack; also that an appendix may perforate or be gangrenous with previous symptoms mild or of short duration. Hence decision not to operate immediately in any case of appendicitis should be arrived at only when the patient can be watched continuously and operated upon without delay, and is not treated in any other circumstances.

Complications and Sequelæ.

1. APPENDIX ABSCESS. (See above.)
2. GENERAL PERITONITIS. (See above.)
3. SUPPURATIVE PYLEPHLEBITIS.—Inflammation may commence in veins near appendix, resulting in (i) Portal pyæmia, tender and enlarged liver, phenomena of severe sepsis. Usual sequela. Always fatal. (ii) Subphrenic abscess. (iii) Occasionally thrombosis, partial or complete, of superior mesenteric vein with gangrene of gut. Initial attack of appendicitis often slight or overlooked. Diagnosis difficult and mortality high.
4. SUBPHRENIC ABSCESS.—Usually from tracking of pus. Moderate irregular prolonged temperature, increased pulse rate, symptoms of sepsis, frequently signs at base of right lung (*see* SUBPHRENIC ABSCESS).
   Also abscess in pelvis and other sites.
5. COLITIS.—If present with onset may persist for long periods.
6. RECURRENT ATTACKS.
7. ADHESIONS.
8. GENERAL SEPTICÆMIA.—Occasionally.
9. HÆMORRHAGE.—Occasional, e.g. perforation of internal iliac artery.

Post-operative Sequelæ.—

(1) **STRANGULATION OF GUT.**—By bands and adhesions. Signs of intestinal obstruction. Occasionally within few days of attacks. Immediate laparotomy necessary.

(2) **SEPTIC COMPLICATIONS.**—*See* COMPLICATIONS. In cases progressing unsatisfactorily examine abdomen, base of lungs, rectum, urine.

3. FÆCAL FISTULA.—Many of the persistent cases are 'tuberculous.' (*see* p. 164)
4. THROMBOSIS OF FEMORAL OR ILIAC VEINS.—Pulmonary embolism may follow.
5. HÆMATEMESIS. This occurs only in severe and septic forms, and is a result of general sepsis and not due directly to disease of the appendix nor connected with the operation. *Hæmaturia and pyuria* may also occur.
6. HERNIA.
7. RECURRENCE OF SYMPTOMS. Specially in chronic form associated with colitis, and not uncommon in the form with dyspepsia.

Various Types of Appendicitis.—In addition to the acute form, the following varieties are met with:

I. 'CHRONIC', 'RECURRENT', AND 'RELAPSING' FORMS.—Two groups of symptoms: (a) *Irregular and mixed symptoms* may be almost constant, viz., vague abdominal pain, loss of appetite, dyspepsia, constipation, often attacks of colic, diarrhœa, and distention. (b) *Recurrent attacks of appendicitis*, varying degrees of pain and tenderness, mainly in right iliac fossa; may be appreciable local resistance and signs often slight; any attack may be followed by security and sequels of acute appendicitis.

Obliterative Appendicitis.—Rarely, with recurrent attacks and chronic inflammation, the mucous lumen becomes obliterated and attacks cease. *Adhesions and thickening* around appendix and cæcum may form dense masses, occasionally hard tumor, simulating chronic proliferative peritonitis. (*See* p. 104.)

II. FORMS ASSOCIATED WITH SYMPTOMS IN OTHER ORGANS.

1. APPENDICULAR DYSPEPSIA.—Chronic irregular dyspeptic symptoms with slight signs of appendicitis, i.e., some pain and tenderness at McBurney's point. Temperature may be normal. Appendicectomy improves condition.
2. SYMPTOMS OF COLITIS. *See* p. 420.

Diagnosis.—Justified by: (1) Sudden pain in right iliac fossa; (2) Fever; (3) Deep tenderness at McBurney's spot; additional symptoms being vomiting, furred tongue, constipation, rapid pulse, rigidity of right rectus, resistance in right iliac fossa, and also, if appendix be distended, superficial tenderness.

DIAGNOSIS FROM:—

1. VARIOUS CAUSES OF PAIN IN RIGHT SIDE.—(1) Renal colic; (2) Biliary colic; (3) Menstrual pains (no fever); (4) Arthritis and pain from hip joint, especially in children; (5) Vertebral disease.
2. DISEASE OF FALLOPIAN TUBES AND PELVIC PERITONITIS.
3. ENTERIC FEVER.—Onset may suggest appendicitis. *Rarely*, appendix ulcerates in third week, and may perforate.

Appendicitis—Diagnosis. *continued*

4 THORACIC DISEASES.—Acute pneumonia, right base; pain at onset may be referred to iliac fossa, especially in children. Also acute pleurisy, intercostal neuralgia.

5 LOCAL PERITONITIS. Due to other causes, e.g. tuberculosis; diagnosis often possible at operation only.

6 PERINEPHRIC ABSCESS.

7 COLITIS AND MUCOUS COLITIS.

8 HYSTERICAL SIMULATION AND HYPOCHONDRIASIS.

9 HERPES ZOSTER.—Rarely.

APPENDIX ABSCESS.—Formation accompanied by (1) Increase in tumour; (2) Constitutional symptoms of sepsis. Rupture marked by shock, collapse, sudden diffuse abdominal pain, progress of general peritonitis.

GENERAL PERITONITIS. Abdominal pain and tenderness, distention and rigidity, pulse rising and usually temperature; also Hippocratic facies, constipation.

Prognosis. Mortality has fallen greatly as result of early operation and adoption of the Fowler position. *In interval appendicitis* practically nil. *In acute attacks* 1 to 2 per cent. with abscess formation 3 to 4 per cent. with peritonitis about 20 per cent., but prognosis varies greatly with extent and severity of infection.

Treatment. *Operation at earliest moment* (see p. 433).

Between time of diagnosis and operation in acute cases place in the Fowler position; water only by mouth; no aperients or enemata; morphia allowable after diagnosis, while awaiting immediate operation.

Interval appendicectomy for one definite attack, however mild; or for repetition of a mild and dangerous attack.

IX. INTESTINAL OBSTRUCTION.

A condition in which the flow of intestinal contents is impeded partially or completely by causes of comparatively local extent. Finally even the passage of flatus is lost. The obstruction may be (1) Acute; (2) Chronic; (3) Acute supervening on chronic.

Causes.*—

CAUSES OUTSIDE THE INTESTINE. (1) Strangulation by bands, adhesions and apertures; (2) Volvulus; (3) Paralytic ileus, rare; (4) Pressure of tumours, rare.

CAUSES IN THE INTESTINAL WALL. (1) Intussusception; (2) Tumours; (3) Strictures; (4) Idiopathic dilatation of the colon.

CAUSES WITHIN THE LUMEN.—(1) Impacted faeces; (2) Gall stones and other foreign bodies.

The common causes of acute and chronic obstruction respectively are not identical. They are as follows:

* By general agreement, the sequelæ of external hernia are not included in the term 'intestinal obstruction'.

ACUTE INTESTINAL OBSTRUCTION.—Common causes (1) Strangulation, (2) Intussusception, (3) Volvulus, (4) Gall stones (uncommon), (5) Chronic forms becoming acute Rarely paralytic ileus (blockage of mesenteric arteries, etc), tumours strictures

CHRONIC INTESTINAL OBSTRUCTION.—Common causes (1) Tumours in wall, (2) Strangulation, (3) Strictures (4) Impacted faeces Rarely pressure of outside tumours chronic intussusception, idiopathic dilatation of colon

General Symptoms.—

A ACUTE INTESTINAL OBSTRUCTION —

1 ABDOMINAL PAIN Early often sudden, severe, at first colicky, then continuous

2 VOMITING Early and constant symptom, repeated, *often copious* *Character* *is most important*, first stomach contents then bilious, finally faecal

3. COLLAPSE Shock at onset progressing to collapse, face pale and pinched low temperature rapid feeble pulse, cold sweat dry tongue and thirst May be in rough

4 CONSTIPATION Absolute for faeces and flatus after few hours, bowel below obstruction sometimes emptying itself by diarrhoea Often distinct but inability to pass flatus

5 ABDOMEN In early stage little change *moderate distention* *Early* variable tenderness often slight no peristalsis Later distention and tympanites depending on cause rigidity and extreme tenderness Tumour rare except in special conditions

PYREXIA Usually absent Temperature often subnormal may rise with peritonitis or remain low owing to collapse

DEATH In 3 to 6 days in absence of operation In later stages, peritonitis present

B CHRONIC INTESTINAL OBSTRUCTION Abdominal attacks similar to acute, but milder and extending over period of months or years severity of symptoms varying and gradually advancing

1 PAIN Colicky intermittent

2 VOMITING Slight or absent may follow food Not faecal

3 GENERAL WEAKNESS Anaemia, wasting, and ill health

4 CONSTIPATION Partial attacks of diarrhoea with mucus, from irritation of scybala above obstruction, sometimes tenesmus, may be morning diarrhoea.

5 ABDOMEN (a) *Distended* (b) *Visible peristalsis* and coils of gut, (c) Often palpable tumour

RECURRENT ATTACKS of severer obstruction symptoms approaching acute form, with marked visible peristalsis; increasing in severity, duration, and frequency.

C ACUTE SUPERVENING ON CHRONIC OBSTRUCTION — The symptoms of acute obstruction with the history and abdominal signs of chronic obstruction

Intestinal Obstruction, *continued*

Notes on Symptoms.—

VOMITING.—The higher the obstruction, the greater is the vomiting.

FÆCAL VOMITING.—Intestinal contents putrefy and thus become "fæcal" above the obstruction; they are not transferred from below. Formed fæces only in hysterical vomiting.

TYMPANITES.—Due to stoppage of blood-supply, not to blocking of lumen. Hence absent in gall-stone impaction and present in thrombosis of mesenteric arteries; marked and rapid in strangulation of large loops, more especially volvulus. In later stages depends on peritonitis.

TENDERNESS AND RIGIDITY.—Not present in early stages in acute form, except in volvulus (from distention). Is due to peritonitis.

TENESMUS.—In colonic obstructions.

PERISTALSIS.—Often best seen after food or abdominal stimulation by flicking or pressure of finger tips.

VARIETIES OF INTESTINAL OBSTRUCTION.

(1) Strangulation of a Loop of Gut.

Commonest cause of acute obstruction (85 per cent.), though infrequent in youth; usually in small gut.

(1) ADHESIONS, BANDS, AND MEMBRANES.—Usually from former peritonitis or result of operations. Meckel's diverticulum may be adherent, usually near navel. Adhesions may form very rapidly, and cause obstruction within a few days of appendicectomy and similar operations.

(2) PERITONEAL POUCHES AND INTERNAL HERNIÆ.—All rare. Strangulation in (i) Foramen of Winslow, (ii) Peritoneal pouches.

(3) DIAPHRAGMATIC HERNIA.—Crura may be (a) Congenital, (b) Acquired, by stabs, crushes, etc. Very rare on right, owing to liver. Hernia usually enters left pleura, rarely mediastinum. *Contents* nearly always stomach. *Physical signs* resemble pneumothorax, but usually borborygmi and frequent variations in the signs. *Symptoms*, vomiting, eructations, etc., often dyspnœa. *Diagnosis* confirmed by giving food and by X-rays and opaque meals. *Treatment* operative.

(2) Intussusception.

"The passing of one portion of intestine into another" (John Hunter).

Pathology.—The upper portion always passes into the lower. The apex remains constant, the tumour growing by invagination of the outer layer. The three layers are: (1) Outer or intussuscipiens; (2) Middle or returning; (3) Inner, entering, or intussusceptum.

TRACTION OF THE MESENTERY.—(1) Bends the tumour, which becomes somewhat curved; (2) Converts lumen at apex

into a slit, easily occluded; (5) Obstructs the vessels, hence œdema, inflammation, adhesion between layers, and gangrene.

MULTIPLICITY.—Very rarely more than one tumour.

Clinical Types. *Acute* and *chronic*. Chronic type has special features and is referred to separately at the end of the section.

Etiology.

FREQUENCY.—Is the usual cause of acute obstruction in children.

AGE.—Under one year, 90 per cent. Rare after age of five.

SEX.—Commoner in male, about two to one female.

RECURRENCE.—Not very rare. In 1 to 2 per cent.

Varieties.—

1. ILEOCÆCAL.—The ileocæcal valve enters colon, often large mass of colon. Typical type, about 50 per cent of all cases.
2. ILEOCOLIC.—Small mass of ileum enters colon.
3. ILEAL.—Confined to small intestine.
4. COLIC.—Confined to large intestine.

Last three types of about equal frequency, more frequent in older patients, especially ileal type.

DOUBLE AND COMPOUND TYPE.—The intussusception may be completely in one or more organs, forming a loop type. Other complex varieties are described.

Origin and Method of Formation. Polypoid growths are occasionally found at apex. Here presumably assists formation of intussusception, as follows. A peristaltic wave is slowly attributed to gut, which is entered by each inner section. Thus first below on thrust, which is followed by small peristaltic waves in train, the normal phenomena which the art tones is longitudinal contraction, relaxation below, contraction above, and a little peristaltic waves. On [illegible] the spasm [illegible] longitudinally, similar actions and action which are [illegible] slowly to the outer over the inner entering layer being pushed. In intussusception, due to abnormal peristalsis and reflex spasm on a portion of wall from ileum.

Exciting stimulus may be a hypertrophied Peyer's patch in a child.

History of previous constipation or diarrhœa occurs in no markedly abnormal proportion of intussusceptions.

Ascending Intussusception. The very few authentic cases are mainly in the colon, where it is known that antiperistalsis may occur.

Symptoms. Patient is commonly a plump, well nourished, healthy infant under one year, generally male.

ONSET.—Sudden.

CHARACTERISTICS.

(1) ABDOMINAL PAIN.—Intermittent. Infant draws up legs and cries during spasm.

(2) VOMITING.—At onset, then ceases. Rarely fæcal.

Intussusception—Symptoms, *continued.*

3. STOOLS.—(i) Tenesmus: (ii) Blood and mucus (from congestion of gut). *Bile absent after few motions* also fecal matter. Quantity small. *Examine per rectum* for presence of tumour and blood on finger, when in doubt.

Physical Signs.—

ABDOMEN.—In early stages appears normal, no distention, palpation often induces spasm

TUMOUR.—Sausage shaped, diameter one inch, length variable, in course of colon, often near left costal margin. Present in 70 per cent.

Diagnosis.—Usually easy on symptoms. Diagnose from colitis.

Treatment and Prognosis. Immediate operation and reduction, after third day usually irreducible. Mortality increases rapidly with delay, being low on first day. Gangrenous gut needing resection is nearly always fatal. With abdominal distention (peritonitis), and child quiet and collapsed, condition is hopeless.

Terminal Invaginations. Found post mortem in children dying from meningitis, peritonitis, or abdominal injuries. *Characteristics.* Usually multiple short in small intestine *ileocecal*, no inflammation, and easily reduced.

Chronic Intussusception. Usually adults or old persons. Due to invagination of malignant or polypoid growth. Type usually *colic or ileal*.

SYMPTOMS.—Chronic obstruction, recurrent irregular attacks of colic and vomiting with bloody diarrhœa or constipation. *Tumour* often palpable. Per rectum, *sphincter relaxed*. Onset may be acute, subsiding into chronic.

TERMINATION. (1) Acute obstruction. (2) Perforation. (3) Occasionally presents at rectum.

DURATION.—A month to a year or more. *Invagination* rarely made.

(8) Volvulus.

A twist of a loop of gut. Due to an abnormally long loop with a long, narrow mesenteric pedicle, twists on long axis, or rarely about another loop. *Sites.* (1) Sigmoid 80 per cent. (2) Cæcum. (3) Occasionally small intestine and other positions.

AGE.—Rare under 40 years.

SEX.—Males 70 per cent.

FREQUENCY.—In adults, is next to strangulation as cause of acute obstruction.

Symptoms and Physical Signs.—*See* ACUTE OBSTRUCTION, p. 435.

SPECIAL SYMPTOMS.—(1) *Abdominal distention and tympanites early and extreme.* Rapid distention is due to occlusion of blood-supply to large loop, and accumulation of gas. Also peritonitis and *gangrene* occur early. (2) Vomiting usually late.

Treatment.—Operation.

(4) Tumours and Strictures.

Common cause of *chronic* obstruction, rare in acute

1 NEOPLASMS OF GUT. —Common, usually carcinoma of sigmoid or descending colon, growth annular or papillomatous

2 STRICTURES Following ulceration of gut In frequency (1) Tuberculosis, (2) Diverticulitis, (3) Syphilis, (4) Dysentery, very rare Typhoid almost unknown

3 EXTERNAL COMPRESSION —Tuberculous peritonitis, pelvic tumours Congenital stenosis is very rare

Symptoms. —*See* CHRONIC OBSTRUCTION, p 435

SPECIAL SYMPTOMS (carcinoma of colon) (1) Morning diarrhœa, (2) Visible peristalsis marked (3) Tumour, (4) Ballooning of rectum and relaxation of sphincter, (5) Rectal hæmorrhage

(5) Causes within the Lumen of the Gut.

1 **Impacted Fæces** See DILATATION OF COLON) Fæces accumulate in colon associated with atony of muscular wall distention may be enormous Occurs at any age especially elderly females Common cause of chronic obstruction *never acute mortality*

Gall-stones. Rare cause of acute obstruction but mortality high never chronic

ETIOLOGY — (1) Age and sex stout elderly females (2) Previous history colic, attacks vomiting and dyspepsia, occasionally but rarely jaundice

PATH OF STONE In cases obstruction stone must have diameter of one inch Usually ulcerates into duodenum through adherent gall bladder if intact or stone may be passed, Passage through bile ducts is recorded

SITE OF OBSTRUCTION Small intestine, ileo-cæcal valve

SPECIAL SYMPTOMS (1) Vomiting constipation, sporadic and *fæcal*, owing to high obstruction (2) Symptoms intermittent passage of fæces and flatus (3) Shock slight at first as mesentery not affected collapse about fourth day Rarely lodges in duodenum, with vomiting of enormous quantities

Mortality from operation high owing to age and frailness of diagnosis and operation due to intermission of symptoms

2 **Other Foreign Bodies.** Very rare Small bodies swallowed accidentally rarely cause trouble Enteroliths fæces phosphates, etc) very rare Bismuth concretions are hypothetical.

(6) Paralytic Ileus

Paralysis of muscular walls may follow any abdominal shock, *viz*, abdominal operations and injuries, peritonitis embolus or thrombosis of mesenteric arteries, rarely paracentesis pneumonia, pleurisy, rarely heart disease Also in hysteria

Embolism of Mesenteric Arteries.

SPECIAL SYMPTOMS Vomiting, constipation, *great abdominal distention*, occurring with heart disease, rarely melæna Peritonitis and gangrene early from poverty of anastomosis.

Intestinal Obstruction, *continued.*

General Diagnosis of Intestinal Obstruction.

Summary of Characteristics.—

A. ACUTE OBSTRUCTION (1) Abdominal pain, (2) Vomiting, copious and finally faecal, (3) Constipation (4) Collapse, (5) 'Doughy' abdomen Temperature usually low

b. CHRONIC OBSTRUCTION (1) Attacks of pain and constipation increasing in severity, duration, and frequency, (2) Abdomen distended, (3) Visible peristalsis, (4) Tumour may be present

Complete diagnosis involves (1) Diagnosis of obstruction from other conditions (2) Of the site of the obstruction (3) Of the nature of the obstruction A careful history of present and previous illnesses is of primary importance

Diagnosis from other Conditions. Often difficult since stimulation of the abdominal sympathetic produces similar results whatever the cause may be

1. ORGANIC OBSTRUCTIONS (a) INTERNAL HERNIA Examine the various rings
2. PERITONITIS Especially *appendicular* (also *ruptured gastric ulcer* Note (1) Abdomen rigid tender and tense (2) (3) Vomiting amount variable and never stercoraceous (4) Pyrexia
3. GASTRO-INTESTINAL IRRITATION Acute enteritis Generally distinguished by diarrhœa From intussusception by less sudden onset below at most no tumour
4. SENSORY STIMULATION OF ABDOMINAL SYMPATHETIC AND ALLIED CONDITIONS Renal and biliary calculi, movable kidney Pelvic cases twisted ovarian tumour (previous or palpable tumour torsion of testis (testis absent) Rarely embolism or thrombosis of mesenteric arteries, i.e. condition of *paralytic ileus*
5. ACUTE HÆMORRHAGIC PANCREATITIS Closely simulates acute obstruction Note Very rapid collapse, feeble pulse, and distention high in abdomen, often pyrexia
6. CONDITIONS ASSOCIATED WITH CONSTIPATION AND SOMETIMES VOMITING Include (i) Enteric (occasionally pneumonia (ii) Tabetic crises (iii) Lead colic (iv) Uræmia (v) Cancer of stomach (vomiting, tumour and constipation) Note (a) Vomiting not faecal, (b) Constipation not absolute (c) No shock

Site of the Obstruction. -

SMALL INTESTINE (1) Vomiting early, copious, and faecal, (2) Distention central, parallel peristaltic coils, ladder pattern (especially if near caecum) (3) Symptoms acute, rapid collapse

* It has been said that a patient with intestinal obstruction vomits into a basin and with peritonitis into a soup dish.

LARGE INTESTINE.—(1) Vomiting later; (2) Distention and peristalsis may be in line of colon; (3) Tenesmus, passage of blood and mucus, suggest colon; (4) Course and collapse often slower.

Nature of the Obstruction.—Often impossible to decide.

IN INFANTS.—Nearly always intussusception.

STRANGULATION BY BANDS, ETC.—Previous operation and attacks.

VOLVULUS.—Elderly males in previous good health; rapid extreme abdominal distention; vomiting later.

TUMOURS.—Previous history of wasting; presence of tumour; peristalsis.

GALL STONES.—Elderly females; previous colic; early copious vomiting but collapse slower.

FECAL OBSTRUCTION.—Faeces often palpable per rectum; colonic distention.

EXAMINATION.—When in doubt examine: (1) Hernial rings; (2) Rectum for tumour, thickening and loss of sphincter; (3) Vagina; (4) In chronic cases, employ opaque meals or enemata (often reveal low obstructions, very valuable).

Treatment.

ACUTE OBSTRUCTION.—Operation without delay.

CHRONIC OBSTRUCTION.—Immediate operation except in the case of *faecal impaction*.

FECAL IMPACTION.—Remove if possible per rectum. Warm olive oil enema followed by soap and water, then remove with spoon. Repeated enemata and careful abdominal massage. Operative treatment gives poor results; removal of mass through incised gut.

Ileus diagnosis, is *a difficult*, keep at rest; gastric *stasis and* [illegible], water in moderate quantity, no enemata except digital to ascertain passage of flatus. To relieve pain hot fomentations, avoid turpentine (to protect skin). Stomach may be washed out, invariably so previous to operation.

No attempt to reduce a volvulus or intussusception by methods other than operation is justifiable.

X. CONSTIPATION.

Delay in evacuation of fæces. Motions less frequently than once in forty eight hours constitute constipation.

Etiology.—The principal factors in peristalsis and in evacuation of fæces are: (1) Abdominal muscles and diaphragm; (2) Intestinal wall, both muscle and mucous membrane and reflex nervous mechanism; (3) Intestinal contents. In organic constipation another factor exists: (4) Obstruction to intestinal contents. The causes of constipation are numerous. They may be classified as: (a) General causes; (b) Local causes, involving mainly one of the above factors. Some overlapping occurs.

A. **General Causes.**—

HEREDITY.—Not uncommon in families.

Constipation—General Causes, *continued*

SEDENTARY LIFE
VARIOUS DEBILITATING CONDITIONS—e.g., (i) Fevers (ii) Anæmia, (iii) Neurasthenia
NEGLECT OF CALL OF NATURE
SENILITY.
DRUG HABITS Especially morphia
HYSTERICAL CONSTIPATION

B. **Local Causes.**—

1. WEAKNESS OF VOLUNTARY MUSCLES (abdominal and diaphragm). Action of these (a) Contractions stimulate peristalsis of gut (b) Contraction during defæcation raises intra-abdominal pressure. Chronic relaxation allows retention and debility of viscera.

   The weakness may be associated with (i) Obesity. (ii) Repeated pregnancies. (iii) Sedentary life. (iv) Visceroptosis. (v) Chronic emphysema, bronchitis, etc. (vi) Senility. (vii) Ruptured perineum, levator ani.

2. AFFECTIONS OF THE INTESTINAL WALL AND NERVOUS MECHANISM.

   AFTER DIARRHŒA AND LAXATIVES. Reflex from mucous membrane deficient and increased absorption of fluid.

   NEGLECT OF CALL OF NATURE. Similar to above.

   DISEASES OF STOMACH. Impaired normal stimulation of stomach to colon.

   ATONY OF COLON. Results from chronic constipation, as Hirschsprung's Disease, etc.

   NERVOUS MECHANISM. Stimulation of the sympathetic system (inferior hypogastric plexus) inhibit peristalsis, the rectum follows.

   LEAD. Acts on sympathetic and causes atony and spasm.

   ENTEROSPASM. Spasm of portion of intestine especially sigmoid flexure. Usually with (i) Chronic mucous membranous colitis. (ii) Neurotic persons. Spasm simultaneously of ileum often causes ribbon-shaped fæces.

3. CHARACTER OF INTESTINAL CONTENTS.

   DIETETIC. Numerous factors, e.g. (i) Diet unsuitable, deficient in articles leaving a residue, also in salts causing peristalsis, e.g. vegetables, fruit. (ii) Diet insufficient, starvation, chlorotic girls. (iii) Fluid insufficient.

   OVERABSORPTION IN INTESTINE. Especially colon. Ingestion and absorption of fluid may be excessive.

4. OBSTRUCTION TO INTESTINAL CONTENTS. Practically identical with causes of intestinal obstruction.

5. INTESTINAL STASIS. Kinks in the intestine described by Lane: (i) In duodenum. (ii) In ileum, ileal kink from dropping of cæcum. (iii) At splenic flexure from dropping of transverse colon. X rays, however, show no delay in fæces passing these kinks.

Types of Constipation. Hurst divides constipation into two groups (1) Slow passage through intestines to rectum (2) Delay and difficulty in emptying rectum (dyschezia). *Normal rate of passage* through intestine is From ingestion of food to cæcum 4½ hours, hepatic flexure 6½ hours, splenic flexure 9 hours, enters pelvic colon 12 hours, enters rectum 15 hours In group (1) delay is in colon the rectum being normally empty delay in passage through small intestine is rare in constipation except with weak abdominal muscles In group (2) passage to pelvic colon is normal or at increased rate The delay is in emptying *rectum*, which *constantly* contains hard feces In this group, when uncomplicated purgatives are of little use and enemata are indicated The factors of both groups not uncommonly co-exist (Hurst)

Symptoms. General moderate impairment of functions Health often fair with chronic constipation

APPEARANCE Complexion muddy, conjunctivæ stained; slight icteric tint not uncommon

ALIMENTARY SYSTEM Tongue furred, appetite poor breath often heavy

EVACUATIONS Infrequent in most cases hard and often scybalous much straining [illegible]

DIARRHŒA [illegible] Rarely in very severe constipation [illegible] masses of feces may simulate [illegible] diarrhœa with mucus [illegible] result

GENERAL SYMPTOMS Headache [illegible] concentration mental depression [illegible] especially with full rectum

ABDOMEN Either normal retracted or distended Distention usually from gas [illegible] Characteristic of this [illegible] easily put on pressure [illegible] extremely rare in cæcum

RECTUM Usually contains hard feces May be empty

PAIN Results from (1) Irregular intestinal contractions (2) Pressure on nerves (3) Frontal left thigh from pressure on anterior crural nerve (4) Lack of thigh or hip pain (sciatica) from 3rd sacral from rectum pressure on 3rd, 4th, or 5th sacral nerves

Sequelæ, Complications, and Remote Effects. These occur as a result of

1 GENERAL ILL HEALTH Boils, acne, anæmia, etc
2 LOSS OF INTRA-ABDOMINAL PRESSURE Hernia, hæmorrhoids, apoplexy, prolapsus
3 IRRITATION OF INTESTINAL MUCOUS MEMBRANE Diverticulitis and pericolitis
4 ACCUMULATION OF FECES —Dilatation of intestines, ultimate form as Hirschsprung's disease Possibly volvulus Nocturnal emissions

Numerous conditions have some association e g, gall stones, mucous membranous colitis. Relation of appendicitis is doubtful Rupture of gut while straining is excessively rare.

Constipation, *continued.*

Diagnosis.—Opaque meals and X rays of great assistance in discovering cause. To estimate time in passage through intestines, give charcoal, and watch for appearance in stools.

Treatment.—

GENERAL METHODS.—

HABIT.—A daily morning stool. Short rest after breakfast often permits stimulus to develop.

EXERCISE.—Walking, etc., or exercises with special apparatus.

MASSAGE AND ELECTRICITY.

BATHS AND HYDROTHERAPY.

DIETETIC.—A sufficient mixed diet containing residue (cellulose) and salts.

SPECIAL ARTICLES.—Porridge, wholemeal bread, fruit of most kinds, apples, prunes, figs, oranges, vegetables.

FLUIDS.—Glass of water half an hour before meals, especially breakfast. Many 'aperient waters' very good.

ENEMATA.—Efficient and harmless over long periods are soap and water enemata and glycerin enemata (ʒiij or with equal volume of warm water). Soap is irritating in some cases and by causing spasm may make constipation worse. Plain water, or water and salt (ʒj to pint), never has this effect. In severe acute constipation, inject olive oil (ʒiv) overnight, and soap and water in morning.

DRUGS.—Numerous drugs are in use. Individuals usually find which suits them best.

XI. VISCEROPTOSIS.

(Enteroptosis. Splanchnoptosis. Glénard's Disease.)

A condition characterized by abnormal descent and mobility of the abdominal contents, accompanied either by irregular symptoms, often neurasthenic, or by none at all.

Symptoms.—Two important groups of cases: (1) Condition follows repeated pregnancies, or ascites, often no symptoms. (2) In young persons, usually thin, lemon-tinted females, in whom enteroptosis is associated with symptoms of neurasthenic character.

The symptoms may be grouped as:—

NEURASTHENIC.—General lassitude, pains in back.

DYSPEPTIC.—Constipation marked, anorexia, and dyspepsia.

CIRCULATORY OR VASOMOTOR.—Faintness, flushing, abdominal throbbing.

Physical Signs.—Abnormal mobility and low position of various viscera. Abdominal wall thin and lax. Pulsations distinct. *Stomach* and *kidneys* are most noticeable displacements. Most neurasthenics show these signs to some degree.

1. STOMACH (GASTROPTOSIS).—Frequent. Greater curvature below, or at least at level of umbilicus; *lesser curvature must be manifest in epigastrium* for diagnosis of gastroptosis;

peristalsis may be present Pylorus at level of umbilicus Splashing common Condition exhibited better after inflation with gas (*see* p 390)

X RAYS APPEARANCES AFTER OPAQUE MEAL —Form and position vary greatly Descent of lesser curvature is essential feature Pylorus little altered in position, and thus is above body, producing 'water trap' stomach Tone may be good i e tubular shape retained but dilatation often occurs and alters appearances (*see* DILATATION OF STOMACH)

2 KIDNEY (NEPHROPTOSIS) Rarely absent in enteroptosis Usually right kidney or both (*see* MOVABLE KIDNEY)

3 DISPLACEMENT OF TRANSVERSE COLON (COLOPTOSIS) *Often palpable as firm cord or sag at level of umbilicus* Lesser descent of hepatic and splenic flexures causes V shaped loop and kinking (Glénard)

4 DISPLACEMENT OF LIVER Not so common Owing to [illegible] liver [illegible] state anterior inferior edge moving backward [illegible] displacement Glénard examines for prolapsed liver as follows left hand grasps loin with fingers behind and thumb in front right hand presses below umbilicus forcing abdominal content under liver thus rotating edge forward against abdominal wall [illegible] becomes palpable to thumb

5 DISPLACEMENT OF SPLEEN Unusual but occasionally extreme

6 DESCENT OF THE HEART Rarely

In addition to the above the following may be noted

DIAPHRAGM In position of inspiration movement slight or absent Respiration may appear thoracic

ROOT OF MESENTERY [illegible] shorter than normal

DUODENUM Traction of suspensory mesentery causes dilatation and hypertrophy Duodenum and jejunum may [illegible]

PYLORUS Near level of umbilicus Often kinked

GALL BLADDER Backward rotation of liver brings gall bladder almost vertical instead of at [illegible] angle Traction of prolapsed duodenum further impede passage of bile

Pathogenesis. The viscera are normally held in position by intra abdominal pressure maintained by muscles of the abdominal wall the functions and action of suspensory ligaments and presence of fat being slight in normal circumstances Fall of intra abdominal pressure is generally accepted as direct cause of visceroptosis Such fall is mainly due to deficiency in the action of the abdominal walls The abdominal muscles may be affected by (1) Distention during pregnancy and subsequent sudden reduction of abdominal contents after parturition, especially if repeated (undoubtedly important) (2) Rapid emaciation (3) Atony disturbance of innervation, separation of recti stretching of perineal floor, etc The erect posture of man is an additional factor The pressure may also be affected by diminution in calibre and contents of intestines, especially colon (Glénard), or by rapid loss of fat.

Visceroptosis—Pathogenesis, *continued*

Some authorities consider that other factors are necessary in addition to such fall of pressure, viz.: (1) *Weakness of suspensory ligaments* (*Glénard*). Descent of right colic ligament considered the initial phenomenon. (2) *Abnormal descent of diaphragm* (*Keith*). The diaphragm is found to be low and in position of complete inspiration, connected with errors in mode of breathing and weakness of crura. Keith ascribes little influence to suspensory ligaments.

Displacement of organs may also result from increased intra-abdominal pressure (e.g., tight lacing) but this is not true visceroptosis.

Explanation of Symptoms. The muscles of the abdominal wall form part of a compensatory mechanism in the abdomen for maintaining the blood pressure constant with changes in position. If mechanism lenient, blood stagnates in the splanchnic vessels and patients "bleed into their own abdomen." Hill, especially on assuming erect posture from lying; hence symptoms of faintness, palpitations, abdominal throbbing, and vasomotor disturbances. Immobility of the diaphragm assists this stagnation. Constipation and dyspepsia are dependent on kinks of colon, displacements of pylorus and duodenum, and deficient passage of bile.

Treatment.—Indications are:

1. MECHANICAL SUPPORT TO ABDOMINAL WALL. Very important and efficacious. Well-fitting belt or bread-trays (to "fit chest") of adhesive plaster attached to thighs front and back and extending across abdomen and lower spine, perhaps two from each side.
2. TREAT NEURASTHENIA. Weir Mitchell treatment if necessary.
3. TREAT CONSTIPATION AND DYSPEPTIC SYMPTOMS. Onset of symptoms often dates from illness and loss of flesh and patient should be fattened.

XII. DILATATION OF COLON.

Occurs in *four forms*, depending on the cause:—

(1) GASEOUS TYMPANITES. Common, rapid and temporary; painful. No obstruction necessary. Small intestine also usually affected. In acute abdominal and certain febrile conditions it may be extreme, affecting heart and lungs. (See TYMPANITES.)

(2) FÆCAL CONCRETIONS. Common. Usually aged females, especially insane. Often stony consistency. Colon thin, no muscular hypertrophy. *Sequelæ*: chronic and acute obstruction, ulceration, perforation colitis. Foreign bodies extremely rare in colon. (See CONSTIPATION, and INTESTINAL OBSTRUCTION.)

(3) ORGANIC OBSTRUCTION.—Usually carcinoma. Colon may almost fill abdomen: muscular hypertrophy (mainly circular

fibres) and dilatation. *Sequelæ*: obstruction, ulceration, perforation, colitis. (*See* INTESTINAL OBSTRUCTION.)

(4) IDIOPATHIC DILATATION OF COLON (HIRSCHSPRUNG'S DISEASE). —*See below.*

HIRSCHSPRUNG'S DISEASE.

Etiology.—In children and young adults; later ages very rare. Commoner in males. *Duration*, several years.

Morbid Anatomy.—Site: sigmoid, and occasionally entire colon. May fill abdomen. Coils twisted. Enormous dilatation, with *muscular hypertrophy*, circular and longitudinal layers. Inflammation of colon frequent. Colonic contents of muddy consistency, and hard concretions. Small intestine collapsed. Wasting, but otherwise body healthy.

Pathogenesis.—Unknown. May vary in different cases. Principal theories:—

(1) LONG SIGMOID FLEXURE AND MESOSIGMOID.—Found occasionally. Possibly kinking results.

(2) CONGENITAL ORIGIN.—Cause unknown.

(3) NARROWING OF LOWER END OF COLON. Very rarely present; certainly not usual origin.

Noticeable facts are: (i) Muscular hypertrophy present; (ii) No obstruction; (iii) Consistency of contents, and incomplete constipation.

Origin possibly resembles that of IDIOPATHIC DILATATION OF ŒSOPHAGUS (*see p. 399*).

Symptoms.—Characteristic:—

1. CONSTIPATION.
2. PROGRESSIVE DISTENTION OF ABDOMEN.
3. PAIN.—Attacks of pain, increasing with distention. Vomiting not marked, often absent.
4. DIARRHŒA.—Gives temporary relief, with reduction of abdomen. Diarrhœa spontaneous or produced by enemata or drugs. Then condition recommences, and abdomen gradually distends.

Physical Signs.—Great abdominal enlargement. Costal arch wide. Colon may be recognizable in left and upper abdomen: during colicky attack more distinct, even separate coils and peristalsis may be recognizable.

Diagnosis.—Usually simple: (1) Age; (2) Constipation; (3) Progressive distention. Sigmoidoscope and X rays after opaque meal and enema confirm.

Progress and Prognosis.—Progresses, in absence of treatment, to acute obstruction or perforation. With treatment, medical or surgical, prognosis remains grave. Death sometimes unexpectedly.

Hirschsprung's Disease, *continued*

Treatment.—

1. MEDICAL.—*Indication is to keep the colon empty* If it is full, empty with enemata When colon has been emptied (i) Enemata, (ii) Massage to abdomen, and general treatment of chronic constipation, (iii) Liquid paraffin and aperients It is necessary to maintain the treatment for a period of several years.
2. SURGICAL.—(i) Complete resection of colon is the most successful method, (ii) Appendicostomy and colonic washes, (iii) Colostomy and colotomy with removal of contents mortality very high.

XIII. MISCELLANEOUS CONDITIONS.

1. DIPHTHEROID ENTERITIS.

(*Croupous Enteritis Secondary Membranous Enteritis*)

Rare Of pathological interest only Not caused by Klebs Loeffler bacillus

OCCURRENCE —

1 SEVERE. ACUTE INFECTIONS Septicemia pneumonia enteric
2 TERMINAL —In chronic wasting diseases cancer nephritis cirrhosis of liver
3 INORGANIC POISONS Arsenic mercury, lead acute

SYMPTOMS Nothing characteristic Diarrhœa if from poisons, severe, in other cases slighter

2. PHLEGMONOUS ENTERITIS.

Similar to but even rarer than, phlegmonous gastritis

VARIETIES

1. PRIMARY Duodenum commonest
2 SECONDARY From intestinal obstruction

SYMPTOMS —As in peritonitis, or intestinal obstruction

DIAGNOSIS —Only at operation or autopsy

3. INTESTINAL SAND.

OCCURRENCE In two forms (1) False, (2) True.

1 FALSE.—Residue after eating certain fruit, especially pears No importance Also in hæmophilia
2. TRUE.—Chiefly seen in muco-membranous colitis Consists of (1) Organic matter, 30 per cent, (2) Inorganic matter, 70 per cent Mainly calcium phosphate No cholesterin Mode of formation unknown.

4. DIVERTICULITIS.

See CHRONIC PERITONITIS, p. 487.

5. AFFECTIONS OF THE MESENTERY.

1. **Hæmorrhage.** Occurs in (a) Aneurysm abdominal aorta, superior mesenteric artery (b) Malignant type of specific fevers e.g., small pox (c) Idiopathic. Also in hæmophilia. Associated with hæmorrhage into retroperitoneal tissues and pancreas.

SYMPTOMS. Acute intestinal obstruction.

2. **Embolism and Thrombosis.** Embolism occurs in arteries from mitral stenosis or infective endocarditis. Thrombosis in veins (a) Secondary to sepsis in appendicitis, pylephlebitis, etc. (b) Primary in cirrhosis of the liver, syphilis, allied conditions.

RESULT. Infarction of the mesentery, terminating in gangrene of intestine supplied, or perforation and peritonitis. In the case of the superior mesenteric vein, chronic intestinal [illegible]

SYMPTOMS.

Acute. [illegible] stools with blood, hæmatemesis, [illegible]

Chronic. May be [illegible]

DIAGNOSIS. Is [illegible] only [illegible]

TREATMENT. Operative, [illegible]

3. **Disease of the Mesenteric Veins.** Suppuration common in pylephlebitis.

4. **Disorders of Chyle Vessels.**

CHYLOUS ASCITES. [illegible]

CHYLOUS CYSTS. See [illegible] THE PERITONEUM, p. [illegible]

*CHYLANGIOMATA. [illegible]

5. **Cysts of the Mesentery.** See [illegible] IN THE PERITONEUM, p. 552.

6. CŒLIAC DISEASE

(*Coeliac Infantile, Coeliac Paralysis, Coeliac Infantilism*)

A condition of infancy, in early [illegible] with defective absorption of fat from the intestine, and the following characteristics: passage of loose, bulky, [illegible] abdominal enlargement, emaciation, and retardation of development.

General Description.—

ONSET.—Usually in early childhood, between age of 1 and 5 years.

MAIN CHARACTERISTICS.—

1. STOOLS.—Loose, pale, bulky, frothy, and offensive. Amount may be enormous. Number of motions variable.
2. ABDOMEN.—Distended usually, soft, flaccid.
3. EMACIATION. *Development retarded.* In those surviving childhood, may be definite infantilism.

VOMITING.—May occur, especially if child is overfed. Flatulence common.

Cœliac Disease, *continued.*

PROGRESS.—Remissions usual; acute and quiescent periods. Prognosis poor, and in severe forms recovery rare.

LATE RICKETS occasionally occur.

Chemical Characters of Stools.

1. *Fat* forms over 30 per cent (may be 40 or 50 of dried fæces in acute stage). Is excessive even in quiescent period. (Normal is about 25 per cent.)
2. The fat is mainly split fat, i.e., acted upon by pancreatic juice. Forms over 80 per cent of total fat.
3. Amount of bile doubtful whether this is deficient.

Theories of Origin. Excessive loss of fat in the stools is undoubtedly the cause of the symptom. Origin of loss disputed:—

1. ENTERITIS. Intestinal alteration is recorded at autopsy, but improbable that this is primary factor.
2. PANCREATIC DEFICIENCY. Suggested by appearance of stools.

   Against:
   a. No defect in fat digestion.
   b. No true steatorrhœa or presence of liquid partially split fat.
   c. No definite azotorrhœa.
   d. No changes in pancreas at autopsy.
3. DEFECTIVE EXCRETION OF BILE SALTS (Miller). A reasonable but unproved theory.
4. PRIMARY DEFECT OF FAT ABSORPTION BY INTESTINE. Either truly primary or secondary to with-drawal of bile salts, is most probable explanation.

Treatment. In acute period, [illegible] diet. For [illegible] suggested by Miller, [illegible] sodium taurocholate and glycocholate [illegible] in water [illegible]. In quiescent periods, attempts should be made to gain fat. No ordinary drugs are of value.

(To Cœliac original [illegible] *Hepatic*, [illegible] also included adult cases from the tropics which suggest sprue or other tropical diarrhœas.)

7. SPRUE.

(*Tropical*)

An inflammatory condition of the alimentary tract occurring in tropical climates, characterized by soreness of the tongue and mouth, chronic diarrhœa, wasting, and anæmia, with tendency to remissions and relapses.

Etiology.—Confined to hot climates. Never epidemic, not contagious. Mainly in Europeans; no obvious connection with diet. Usually after long residence; rarely in 1 to 2 years or less. Aided by dysentery and debilitating conditions.

No cause known. Moulds (*Monilia*) often present, but probably secondary invaders. Pancreatic deficiency suggested, but no constant changes found.

Pathology. Commences as acute catarrhal inflammation of alimentary tract followed by ulceration. Small intestine usually severely affected but no portion immune and mouth almost early and constantly involved. In later stages atrophy of mucous membrane in colon and mucous cysts. Liver smaller than normal. Mesenteric glands enlarged.

Symptoms.

ONSET. Gradual or sudden, [illegible] dysentery or diarrhœa. Stomatitis constant, may be [illegible] before diarrhœa. Remissions and recurrences usual, duration frequently [illegible]. Entire alimentary tract finally affected.

CHARACTERISTIC SYMPTOMS.

Stomatitis. Long [illegible] painful catarrhal [illegible] ulceration. Subsequently atrophy and [illegible].

Diarrhœa. Stool greyish white, [illegible] *Fat* [illegible] large amounts [illegible] but [illegible] returned [illegible].

Wasting.

LATER SYMPTOMS.

Anæmia. In [illegible] later stages [illegible] anæmia [illegible].

Dysentery. [illegible].

Nervous. [illegible].

Hands and feet cold, [illegible] and [illegible].

Treatment. *Mouth and Stomach.* [illegible] with [illegible] point [illegible] for at least 4 weeks [illegible] in [illegible] Sodium bicarbonate [illegible] with a few drops of [illegible] 3 times daily and continue as long as [illegible] effects.

If [illegible] very [illegible] days [illegible].

Mouth must be kept [illegible] stomatitis, a simple mouth wash.

For persistent diarrhœa [illegible] BACILLARY DYSENTERY. If constipation, liquid paraffin.

Check acute symptoms and then return to drugs until cured for a year.

Treatment is prolonged but if obtainable as above, usually successful, much atrophy of mucous membranes of alimentary tract is permanent after long attacks and recurrences and subsequent care necessary.

CHAPTER LXXVII.

DISEASES OF THE LIVER.

I. JAUNDICE.

(*Icterus.*)

A condition characterized by staining of the skin and mucous membranes and tissues by bile pigment, and usually by its presence in the body fluids. Jaundice is a symptom and not a disease. Two principal groups recognized: (1) *Obstructive*; (2) *Toxic or hemolytic*.

Mechanism of Jaundice.

(1) OBSTRUCTIVE JAUNDICE. Increase in the bile capillaries rises; finest capillaries dilate, rupture into lymphatics and bile reaches blood via thoracic duct; possibly also direct into blood capillaries (*vide*).

(2) TOXIC OR HEMOLYTIC JAUNDICE. Mechanism less clear. Formerly all jaundice divided into hepatogenous (or obstructive as above) and hematogenous, or non-obstructive. In latter type bile was supposed to be formed in the blood from freed hemoglobin. Now known that bile is only formed in liver cells. Now recognized by many a toxic, hemolytic or infectious intrahepatic hepatogenous jaundice. Factors probably concerned:

i. Increased formation with resulting increase bile pigment essential factor.

ii. Inflammation and swelling in smallest bile capillaries (cholangitis) due to cause of condition here called [illegible] by the action of regolithic bile and increased viscosity of bile. Hence capillaries dilate at end and rupture as in obstructive form. Lymphatic jaundice supports possibility also of direct absorption into blood as cholangitis is absent.

iii. Necrosis of liver cells focal or diffuse, usually and possibly always present in varying degrees.

Hepatic Efficiency: Lævulose Test. Ingestion of lævulose 50 to 50 grm. has no effect on blood sugar in normal persons. With liver inefficiency a rise occurs resembling normal blood sugar curve after glucose; the height and duration of rise appear to be proportionate to degree of inefficiency (Spence and Scott).

1. OBSTRUCTIVE JAUNDICE.

Causes.—Obstructions arising in the lumen, in the walls, or external to the common bile or hepatic ducts.

FOREIGN BODIES IN THE DUCTS.—Gall-stones, parasites.

INFLAMMATORY SWELLING OF MUCOUS MEMBRANE OF DUCTS.—Often extension from duodenum, e.g., ACUTE CATARRHAL JAUNDICE (*see* p. 459)

TUMOURS OF THE DUCTS.

STENOSIS OF THE DUCTS.

EXTERNAL PRESSURE ON THE DUCTS.—Especially: (*a*) Tumours of liver, pancreas, and stomach, occasionally kidney, etc.; (*b*) Glands in liver fissures.

Jaundice less severe and constant in following:—

CIRRHOSIS AND DIFFUSE DISEASES OF LIVER.—Occluding small ducts.

KINKING OR TORSION OF DUCTS.—A doubtful cause: visceroptoses, pregnant uterus, faecal masses.

Symptoms.—Result from (*a*) Presence of bile in blood; (*b*) Absence of bile from intestine; (*c*) Hepatic toxaemia from disturbed function; and (*d*) Cause condition.

ICTERUS.—Affects all tissues except central nervous system. Earliest in conjunctivae. Persists often one or more weeks after absence of bile from urine. Often overlooked at night. Colour: light yellow to intense green, green bronze.

BILE PIGMENTS IN URINE AND OTHER SECRETIONS.—*Urine*: green tint, may precede icterus; usually contains albumin, and *bile stained hyaline casts*. Milk, saliva, and sputum discoloured (except last if pneumonia present).

CLAY-COLOURED STOOLS.—Often large and offensive. *Colour* due to (*a*) Absence of bile pigment urobilin, stercobilin; (*b*) Excess of fat. *Odour* due to higher fatty acids. Excessive fermentation, formerly attributed to absence of the supposed antiseptic action of bile, usually not present.

CONSTIPATION.—Bile aids intestinal peristalsis. Diarrhoea if much fermentation. Anorexia, furred tongue, and gas disturbance rarely absent.

ITCHING.—Often severe in chronic conditions.

OTHER SKIN CONDITIONS, such as eczema, urticaria, boils, occasionally hæmorrhages, rarely xanthelasma.

NERVOUS SYSTEM.—Depression and irritability are marked; occasionally melancholia.

Other noticeable symptoms:

SLOW PULSE.—In early jaundice only, not extreme; often absent.

BLOOD.—Serum bile tinged (recognition simple). Coagulation time prolonged.

HÆMORRHAGES.—In acute and chronic cases, tendency to hæmorrhages, e.g., at operations; also telangiectases, purpura.

XANTHOPSIA, OR YELLOW VISION.

XANTHELASMA.—Rare. Yellowish plaques or areas: commonest on eyelids, very rarely diffusely on body (? deposits of cholesterin).

CHOLÆMIA.—*Often rapidly fatal.* In acute febrile types, or occasionally terminating chronic jaundice. Coma or delirium, rapid pulse, dry tongue, 'typhoidal state'. Cause uncertain: probably loss of detoxifying function of liver cells. Similar condition occurs in hepatic cirrhosis in absence of jaundice.

Obstructive Jaundice, *continued.*

Notes.—

LIVER, GALL-BLADDER, AND SPLEEN.—The question of enlargement depends on the cause of the jaundice.

FAT IN FÆCES IN JAUNDICE.—Mainly as *fatty acids*, unless pancreatic secretion is also absent (*see* p. 484).

ERYTHROCYTES in jaundice are abnormally resistant to hæmolysis (except in acholuric family jaundice), measured by action of hypotonic salt solutions: possibly compensatory to bile salts, which are strongly hæmolytic *in vitro*.

BILE SALTS.—Present in blood only in early stages. Slow pulse is ascribed by some to their stimulation of vagus, by others to action on myocardium or cardiac ganglia.

2. TOXIC OR HÆMOLYTIC JAUNDICE.

Causes.—

1. DIRECT POISONS.—(i) Organic, e.g., trinitrotoluene, toluenediamine, tetrachlorethane, chloroform; (ii) Inorganic, e.g., phosphorus, rarely arsenic, arsenobenzol preparations; (iii) Hæmolysins, e.g, snake-venom.
2. SPECIFIC INFECTIONS AND TOXÆMIAS.—(i) Toxæmia of pregnancy; (ii) Pyæmia and septicæmia; (iii) Malaria, (iv) Yellow fever; (v) 'Epidemic jaundice' (Weil's disease). Occasionally in syphilis, relapsing fever, typhus, and typhoid (very rare).
3. CERTAIN CHRONIC HÆMOLYTIC CONDITIONS.—Acholuric family jaundice; pernicious anæmia (marked jaundice rare).

Symptoms.—Vary greatly with the cause, constitutional symptoms resulting from this usually being very severe, and direct symptoms of jaundice little marked. Degree of jaundice often slight in proportion to general symptoms. 'Typhoidal state', rapid pulse, dry tongue, hæmorrhages, and death common.

STOOLS.—Contain stercobilin, and are not always 'clay-coloured'.

URINE.—Bile pigment usually slight; may be absent, but urinary pigments increased owing to absorption of stercobilin (identical with urobilin) from intestines.

II. ICTERUS NEONATORUM.

Many types. Severer forms (except deformities) are akin to hæmorrhagic diseases of the new-born.

Physiological Jaundice.—

FREQUENCY.—About 50 per cent of infants. *Onset* within first four days.

JAUNDICE.—Mild; rapidly reaches maximum; duration about two weeks. No symptoms. Conjunctivæ often escape. Bile in urine rare. Liver and spleen not enlarged. Never fatal. No treatment necessary.

CAUSE.—Large destruction of erythrocytes after birth. Possibly aided by bacilli, from previously sterile intestine, causing mild cholangitis.

Severe Forms.—

CONGENITAL ABSENCE OF THE BILE DUCTS (*see* p. 466).

CONGENITAL SYPHILITIC HEPATITIS (*see* p. 270).

SEPSIS.—Usually phlebitis of umbilical vein; severe constitutional symptoms; suppuration at navel; hæmorrhages common. Recovery rare.

Rare Forms.—

EPIDEMIC JAUNDICE OF INFANTS.—Jaundice, diarrhœa and hæmaturia; may be hæmoglobinuria. Is same as Winckel's disease, epidemic hæmoglobinuria.

FAMILIAL JAUNDICE.—Successive infants become jaundiced; high mortality. Mother may also be jaundiced.

ACHOLURIC FAMILY JAUNDICE (Congenital Splenomegalic Jaundice).—May date from birth.

VIII. ACUTE YELLOW ATROPHY.

A rare acute condition characterized pathologically by necrosis of the liver cells and diminished size of the liver, and clinically by jaundice, nervous symptoms, toxæmia, small liver, and high mortality. Always secondary, and is not a clinical entity.

[*Icterus gravis.*—Term applied to jaundice when associated with severe toxæmia, e.g., in yellow fever.]

Etiology.—

AGE.—20 to 40 years usually. Rarely in children.

SEX.—Females preponderate (from influence of pregnancy).

PREDISPOSING CAUSES.—Condition is always secondary, and the causes are practically those of hæmolytic jaundice (*see* p. 451).

Notes on Causes.—

PREGNANCY.—Accounts for at least 30 per cent. of all cases. Usually latter half of pregnancy; occasionally in puerperium; very rare before 4th month. The common foci of necrosis in liver in pregnancy are of same type, but of less extent.

PHOSPHORUS.—Liver usually enlarged and fat very excessive; but in less acute poisoning of some duration, liver is identical with other forms, fat probably being absorbed.

SYPHILIS.—Secondary syphilitic hepatitis rare cause. Probably intercellular fibrosis, with final necrosis of cells. Spirochætes said to be absent. Duration somewhat longer than other forms.

Pathogenesis.—Obscure. Probably various causes and toxins, e.g., 'epidemic jaundice' cause similar necrosis. Association with 'acidosis' definite, but not invariable, and relationship uncertain.

FLEXNER'S THEORY OF 'AUTOLYTIC NECROSIS'.—Some toxin kills the liver cells without destroying their autolytic ferments, which then cause necrosis of the dead cells.

Acute Yellow Atrophy, *continued.*

Morbid Anatomy.—

LIVER.—*Size greatly reduced.* Weight often 20 to 30 ounces or less. Greenish-yellow colour. Flabby. Capsule wrinkled and strips easily: below, may be hæmorrhages. *On section*: yellow and red areas, mottled appearance.

YELLOW AREAS.—Colour due to bile. Contain fat and necrosed cells. *Histology*: Necrosed cells in all stages, hæmorrhages between cells; condition commences in intermediate zone of lobule; cholangitis of small bile-ducts, with increase in number.

RED AREAS.—Later stage of above, fat and necrosed tissue being absorbed. Fibrous tissue and capillaries alone remain (whence colour). Depressed below yellow areas. *Histology*: Often unrecognizable as liver. The longer the duration, the greater is the proportion of red areas.

AMOUNT OF FAT.—Usually somewhat increased, 5 to 10 per cent against normal 3. (In phosphorus poisoning forms 50 to 80 per cent.)

LEUCINE, TYROSINE, and other amino-acids greatly increased: may precipitate as film on cut surface. Origin of leucine and tyrosine probably from liver cell degeneration.

OTHER ORGANS.—Bile-stained, and hæmorrhages numerous. *Kidneys*: epithelial degeneration *Heart*: fatty degeneration. *Spleen*: usually enlarged. *Blood*: fluid, stains endothelium.

Subacute Yellow Atrophy and Liver Regeneration.—In cases of longer duration, raised nodules often present. *Histology*: Consist of cells resembling liver cells, and others in columns suggesting ducts: active karyokinesis and proliferation. Are regarded as areas of 'liver regeneration'. Either: (*a*) Surviving liver cells proliferate, and form new bile-ducts; or (*b*) Interlobular bile-ducts proliferate, and produce 'liver cells'.

Symptoms.—Two stages:—

(1) FIRST STAGE.—Gradual onset: resembling acute catarrhal jaundice, but vomiting frequent. Duration five days or longer.

(2) SECOND STAGE.—Rapid development of severer, and *nervous symptoms. Headache,* muscular twitching, convulsions or delirium passing to coma and death. *Vomiting* intractable. *Jaundice* usually deepens. Abortion if pregnant. *Petechiæ* and hæmorrhages common: skin, mucous membranes, retinæ especially. Cholæmia develops with 'typhoidal state', rapid pulse, dry tongue, etc. Duration two to seven days. Temperature variable: high before death.

LIVER DULLNESS.—Diminishes progressively: even entirely obliterated if liver falls back and distended intestines pass in front.

URINE.—*Amount* diminished. *Bile* present. *Albumin and casts*: usually large quantities. Sugar absent. *Excretion of nitrogen* as in acidosis: (1) Total nitrogen diminished; (2) Percentage of urea low; (3) Percentage of 'ammonia-nitrogen' very high, 20 to 50 per cent; (4) Amino-acids in excess. *Leucine and tyrosine*

usually present, occasionally form precipitate, but may be absent; and presence is not diagnostic of acute yellow atrophy, occurring occasionally in various acidoses.

CONSTIPATION.—Severe. Stools often darkened with blood, and offensive.

BLOOD.—Serum bile-stained. Coagulation delayed. Very fluid.

Diagnosis.—Essential symptoms are: (1) Jaundice; (2) Vomiting; (3) Nervous symptoms; (4) Small liver; (5) Urinary changes.

PHOSPHORUS POISONING.—Distinguished by: (1) Distinct remission between two stages of symptoms; (2) Liver enlarged; (3) Widespread fatty degeneration.

Prognosis.—Mortality very high, especially in pregnancy: less in children. Rarely, improvement and prolongation for weeks, with subsequent death. *Subacute* forms with recovery occur: diagnosis often uncertain.

Treatment.—In pregnancy, the treatment is that of eclampsia. In all forms the *acidosis* must be treated.

IV. AFFECTIONS OF THE BLOOD-VESSELS OF THE LIVER.

1. PASSIVE CONGESTION OF THE LIVER.

('*Nutmeg' Liver. Cardiac Liver.*)

Cardiac 'back-pressure' of any origin causes increased pressure in the efferent vessels of the liver, and hence mechanical congestion, which results finally in pathological changes.

Causes.—

CARDIAC LESIONS.—Especially mitral stenosis.

PULMONARY CONDITIONS.—Emphysema and chronic bronchitis. Interstitial fibrosis of lung. Intrathoracic tumours and aneurysms: very rare cause.

Morbid Anatomy.—

LIVER.—Large, firm, smooth and dark red. *On section*: Surface mottled, i.e., 'nutmeg', due to zones in the lobules: (*a*) Intralobular veins dilated (red centre of lobule); (*b*) In remainder of lobule, cells bile-stained, atrophied, or in fatty degeneration (yellow periphery of lobule).

HISTOLOGY.—Lobule shows: (1) Intralobular veins and their capillaries distended: extent and area vary. (2) Intermediate zone: liver cells compressed, and later atrophy and necrosis: deposit in cells of *brown pigment*, i.e., iron-free hæmatoidin (Prussian-blue test negative). (3) Peripheral zone: often fatty degeneration. Small hæmorrhages between cells.

Connective tissue often increased, but *no red cirrhosis* when uncomplicated. Hepatic veins dilated and walls thickened.

'CYANOTIC INDURATION'.—In later stages, when chronic, liver may become contracted and tough.

Passive Congestion of the Liver, *continued.*

Symptoms.—Dominated by causal disease.

GASTRIC CATARRH, FLATULENCE, ETC. Also when disease is advanced: ascites, slight jaundice, hæmatemesis.

LIVER.—Enlarged: size often varies rapidly, e.g., smaller after hæmatemesis. Tender (measure of severity). *Pulsating liver* test by anteroposterior examination to differentiate from transmitted pulsation.

Diagnosis.—From cirrhosis by: cardiac and lung lesions, liver surface smooth, no distended abdominal veins.

Treatment.—Open bowels freely (salines or calomel). For severe pain: poultices or leeches. Treatment mainly directed towards cause.

2. THROMBOSIS OF THE PORTAL VEIN.

(*Pylethrombosis. Adhesive Pylephlebitis*)

Thrombosis occurring without suppuration: condition *rare.* For *suppurative pylephlebitis, see* ABSCESS OF LIVER, p. 479.

Etiology.—May result from any pressure on, or affection of, portal vein or immediate tributaries:—

CIRRHOSIS OF LIVER.—In 1 to 3 per cent of cirrhosis (any type, including syphilis). Is commonest cause.

CANCER INVADING OR CONSTRICTING VEIN.—Usually liver, pancreas, or stomach.

GALL-STONES.—Invading portal vein.

INFLAMMATION FROM OTHER CAUSES SPREADING TO VEIN.—Inflammation in neighbouring tissues, suppuration in or near liver, gall-bladder, and pancreas.

THROMBOSIS SPREADING FROM TRIBUTARIES. As from splenic vein (from suppuration, infarct, or other disease), or from superior mesenteric vein.

Many rare and occasional causes, e.g., syphilitic phlebitis, phlebosclerosis, calcification of vein; pregnancy; chronic proliferative peritonitis. No cause may be found: often thrombosis elsewhere.

Pathology.—

PORTAL VEIN.—If thrombosis is recent, vein is distended with clot; walls may be sclerosed. Intestines may be gangrenous if superior mesenteric vein involved, especially jejunum (no anastomosis with parietal veins). In chronic forms, clot organizes and vein becomes a fibrous cord or is canalized; collateral circulation is established. Extent of clot varies, may extend from portal vein into tributaries, or vice versa.

LIVER.—Some atrophy and fibrosis, but often very little change. Infarcts not uncommon.

SPLEEN.—Nearly always enlarged.

The clotting possibly results directly from phlebosclerosis; influence of organisms unknown.

Symptoms.—Vary with: (1) Extent and site of clot; (2) Rapidity of formation. Pre-existing abdominal disease usual.

SUDDEN THROMBOSIS.—Sudden symptoms of engorged portal system: hæmatemesis, enlargement of spleen, ascites, melæna. Superficial abdominal veins sometimes distended. Abdominal pain.

LOCAL SYMPTOMS.—Depend on distribution of clot: e.g., symptoms of intestinal obstruction (mesenteric veins thrombosed).

CHRONIC CONDITION.—Signs of collateral circulation. Hæmatemesis often recurrent. Finally fails, with ascites, etc.

Diagnosis.—Rarely possible. Obscured by primary disease.

Prognosis.—Death usually rapid, few days to few weeks. With carcinoma may be several months. With cirrhosis may be a year or more. Recovery occasionally occurs, with several years of life.

Treatment.—Palliative. In chronic forms, elastic bandages to support superficial veins.

V. DISEASES OF THE BILE PASSAGES AND GALL-BLADDER.

1. ACUTE CATARRHAL JAUNDICE.

(*Acute Catarrhal Cholangitis.**)

Jaundice due to obstruction of the common bile duct, resulting from inflammatory swelling of the mucous membrane at its termination.

Etiology.—

AGE.—*Children and young adults* most frequent, but any age liable.

DUODENAL OR GASTRO-DUODENAL CATARRH.—Due to: (1) Indigestion, cold, and exposure, (2) Chronic heart disease, nephritis, portal cirrhosis. Ascribed to inflammation of duodenum extending into duct, but there is no proof of this sequence, and the cause of inflammation is unknown.

Overwork or worry are not uncommonly the only obvious preceding factors.

INFECTIOUS FEVERS.—E.g., pneumonia. In typhoid very rarely. This group possibly is toxic jaundice. Also in 'epidemic jaundice'.

Pathology.—Mucous membrane swollen at termination of duct and in duodenum. Plug of inspissated mucus in ampulla of Vater. Condition rarely extends far along ducts.

Symptoms.—

PREMONITORY SYMPTOMS.—General malaise and gastric disturbance, duration seven to ten days: common, but usually slight. Vomiting occasionally severe.

JAUNDICE.—Bright yellow: often first symptom. Never dark tint of chronic jaundice.

* Cholangitis and angiocholitis are synonyms.

Acute Catarrhal Jaundice—Symptoms, *continued.*

LOSS OF APPETITE.—*Nausea* and vomiting (especially if diet excessive). Headache, furred tongue, and malaise. Symptoms may precede jaundice, and subside as it appears.

TEMPERATURE.—Variable: normal, or 101° to 102°.

SYMPTOMS AS IN OBSTRUCTIVE JAUNDICE.—Bile in urine, clay-coloured stools, constipation, mental depression, itching, slow pulse, bile-tinged serum. No biliary colic, or severe pain. Pains in back and limbs at times.

LIVER.—Often slightly enlarged and tender. Gall-bladder may be palpable: spleen rarely.

Course and Termination.—*Duration,* two to five weeks: colour often fades slowly. Over six weeks' duration suggests carcinoma or gall-stones in adults. Simple catarrhal jaundice never fatal.

Diagnosis.—In young subjects rarely difficult: premonitory malaise; absence of colic, physical signs, and severe symptoms. In older patients, exclusion of carcinoma needs longer observation.

Treatment.—Rest in bed and warmth.

DIET.—Fluids at onset. *Avoid fats.* Progress gradual: (1) Broth, milk, peptonized milk, gruel—for one to three days; then (2) Benger's food, milk puddings, custard, egg-flip, and eggs, (3) Pounded or boiled fish.

BLAND FLUIDS.—Use encouraged. Hot water at onset, especially if vomiting. Then mineral waters, or water with sodium bicarbonate added.

BOWELS.—Free motions, but avoid purging. Calomel, gr. ½ to 2, first evening: then morning salines.

GASTRIC SEDATIVES.—Especially bismuth.

℞ Bismuth. Salicylat.	gr. xv	Tinct. Aurantii	♏ xv
Acid. Hydrocyan. Dil.	♏ iij	Aq.	ad ℥ j

t.d.s.

INTESTINAL ANTISEPTICS.—Salol gr. x. t.d.s. Widely used: action doubtful.

EPIGASTRIC DISCOMFORT.—Hot fomentations.

DURING CONVALESCENCE.—Avoid chills, and heavy diet. Strychnine tonic. Slight jaundice may persist after symptoms subside. Gastric condition is best guide for treatment.

2. CHRONIC CATARRHAL CHOLANGITIS.

Invariably present in chronic obstruction of common ducts from any cause (*see* OBSTRUCTIVE JAUNDICE, p. 452). Apparently never a permanent sequel of acute catarrhal jaundice.

3. SUPPURATIVE CHOLANGITIS.

Purulent inflammation of the bile-ducts, large and small. Gall-bladder but rarely escapes (acute cholecystitis).

Etiology.—Any condition affecting bile-ducts, and thus rendering them liable to bacterial invasion, but all causes *rare* except gall-stones.

1. GALL-STONES.—Cause of 90 per cent. Severest sequel of gall-stones
2. ACUTE INFECTIVE CHOLECYSTITIS.—Spread is rare: possibly cystic duct occluded
3. CANCER OF DUCTS
4. ROUND WORMS. FOREIGN BODIES. RUPTURE OF HYDATIDS.
5. SUPPURATIVE PYLEPHLEBITIS
6. INFECTIOUS FEVERS, e.g., pneumonia and influenza.

Morbid Anatomy.—

COMMON DUCT.—Dilated, often enormously. Walls thick and inflamed

LIVER.—Enlarged. On section, *multiple small abscesses* or multiple yellowish areas in process of suppuration: rarely, single large abscess. Hepatic ducts and tributaries distended with bile-stained pus

GALL-BLADDER.—Usually distended with pus (empyema)

Various adhesions, or fistulæ of ducts or gall bladder into intestines, pancreatitis, pylephlebitis, peritonitis, pleural effusion, and other effects of extension of pus

Symptoms.—*Severe sepsis, with previous history of gall-stones*

ONSET.—Rigors, nausea, great prostration. Temperature variable.

JAUNDICE.—Usually intense, occasionally slight

PAIN OVER LIVER.—Worse on movement (perihepatitis)

LIVER.—Progressive enlargement. Surface smooth. Tender.

Gall bladder usually enlarged. Spleen occasionally. Leucocytosis present. Blood culture various bacteria recorded

PROGRESS.—Rapid emaciation, prostration, and usually death.

Complications.—Numerous from spread of pus and septicæmia: suppurative pylephlebitis, pancreatitis, peritonitis, pleural effusion, endocarditis. When spontaneous recovery, fistulæ and strictures of ducts.

Diagnosis.—

CHARACTERISTICS.—Severe sepsis, jaundice, enlarging liver, history of gall-stones, *symptoms progressive.*

DIAGNOSIS FROM—

1. HEPATIC INTERMITTENT FEVER (*Infective cholangitis*).—Free intervals, with recurrent attacks of syndrome: jaundice, colic, rigors, sweats, and fever (*see* p. 471)
2. PYLEPHLEBITIS.—May co-exist. Symptoms similar. Usually from appendix.
3. TROPICAL ABSCESS OF LIVER.—History of dysentery.

Prognosis.—Mortality high. With liver abscess formation, all fatal. Recovery depends upon evacuation of pus in ducts before involvement of liver, by (1) operation—prognosis fair, (2) fistulæ, and discharge into intestines spontaneously.

Treatment.—Immediate operation, evacuation of pus, and drainage.

4. ACUTE CHOLECYSTITIS.

Acute inflammation of the gall-bladder resulting from action of various bacteria.

Etiology.—

GALL-STONES.—In the gall-bladder or cystic duct; or less commonly, combined with suppurative cholangitis, from stone in common duct. Usual cause.

ACUTE NON-CALCULOUS CHOLECYSTITIS.—From certain infections, e.g.: (1) Typhoid or paratyphoid fevers, bacilli may be isolated ten to twenty years later; (2) Influenza.

Appendicitis may co-exist: relation doubtful.

Degrees of Severity.—(1) Catarrhal; (2) Suppurative; (3) Phlegmonous. The very rare phlegmonous type is referred to separately. Catarrhal and suppurative forms differ in severity, and by presence of pus in latter: intermediate types occur, and differentiation of the form is frequently impossible. Empyema (chronic or simple) of gall-bladder links the two forms; pathologically the suppurative form is always an acute empyema; but the term 'empyema' is more generally applied to the chronic accumulation of pus.

Morbid Anatomy.—

ACUTE CATARRHAL CHOLECYSTITIS.—Gall-bladder distended and tense; walls thickened; mucous membrane congested and covered with mucus, and often ulcerated; contents either serous or turbid sero-fibrinous or bile-stained fluid; gall-stones usually present in gall-bladder or duct; cystic duct often occluded. Adjacent lymphatic glands enlarged. Adhesions to colon, etc., common.

SUPPURATIVE CHOLECYSTITIS.—Changes as above, but more severe: *gall-bladder contains pus.*

Symptoms.—Usually history of previous biliary colic.

AT ONSET.—

1. PAIN.—Severe and paroxysmal. Usually over liver (as in colic). Occasionally in right iliac fossa, or epigastrium
2. TENDERNESS.—Marked. General, and then localizing near 9th rib.
3. JAUNDICE.—Absent.
4. GALL-BLADDER.—Usually palpable.
5. LIVER.—Not enlarged, unless cholangitis present, or Riedel's lobe from previous gall-stones.

Rectus rigid. Hyperæsthesia of eighth and ninth dorsal segments.

CATARRHAL FORM.—Moderate gastric disturbance, and intestinal distention from local peritonitis. Temperature often normal.

SUPPURATIVE FORM.—Signs of sepsis: rigors, rapid pulse, high temperature, prostration. Intestinal distention, and often symptoms of obstruction (vomiting, complete constipation) from local peritonitis: may mask enlarged gall-bladder.

Sequelæ.—

OF CATARRHAL CHOLECYSTITIS.—The sequelæ are subacute or chronic :—

EMPYEMA OF GALL-BLADDER (chronic or simple). *Important.* Acute symptoms subside. Then gradually malaise, anorexia, abdominal pain, and gall-bladder tumour: temperature slight. Due to slow formation of pus.

ADHESIONS.—To stomach, etc.; causing gastric disturbances, often vague.

CHRONIC CHOLECYSTITIS.

OF SUPPURATIVE CHOLECYSTITIS (EMPYEMA).—Sequelæ may be acute :—

1. PERFORATION. —(i) General peritonitis, but adhesions often prevent this; (ii) Local abscess formation, e.g., subphrenic abscess; (iii) Into duodenum, colon, etc. (after adhesions); (iv) May point through the skin.
2. INFLAMMATION SPREADS through wall to neighbouring structures (pericholecystitis).
3. ADHESIONS.—Resulting from spread of inflammation
4. SUPPURATIVE CHOLANGITIS.—Rare, owing to occlusion of duct.

Appendicitis may co-exist. *Intestinal obstruction* may be simulated.

In severe acute catarrhal forms, similar sequelæ may occur.

Diagnosis.—Difficult. Symptoms do not localize lesion. Previous history of gall-stones (or enteric) important. Diagnosis from :—

1. DISEASE OF ABDOMINAL ORGANS NEAR LIVER, e.g.: (i) Perforated duodenal ulcer; (ii) Right acute pyelonephritis (pus in urine) symptoms of these are indistinguishable; (iii) Subphrenic abscess.
2. DISEASE OF RIGHT BASE.—Pneumonia and pleurisy.
3. APPENDICITIS.
4. ACUTE INTESTINAL OBSTRUCTION.—Occasionally.

Prognosis.—In milder catarrhal forms condition usually subsides. In severer and suppurative forms, prognosis depends on early operation; mortality considerable. In chronic empyema, and localized abscess, prognosis good after operation, but death rate not negligible.

Treatment.—In milder catarrhal forms, treatment as for catarrhal jaundice. (*See also* GALL-STONES) (Withhold morphia, which may mask dangerous symptoms.) For suppurative cholecystitis and empyema of gall-bladder, immediate operation.

Phlegmonous Cholecystitis.—Very rare.

Symptoms resemble suppurative form, but of great severity and rapidity: toxæmia extreme, jaundice not uncommon. Gall-bladder swollen, œdematous, and very friable. Rapid sloughing, perforation, and general peritonitis usual: adhesions rare, from short duration. *Treatment*: immediate operation and removal. Mortality very high.

Gangrenous Cholecystitis.—Is a sequel of above.

Diseases of the Gall-bladder, *continued.*

Chronic (Catarrhal) Cholecystitis.—

ETIOLOGY.—Sequel of gall-stones; inflammation either chronic from onset, or follows acute cholecystitis.

MORBID ANATOMY OF GALL-BLADDER.—May be definitely enlarged, and lumen distended with ropy mucus; no bile present. Adhesions common. *Walls* thickened with fibrous tissue; little normal mucosa remains.

Every intermediate grade occurs between this and cholecystitis obliterans.

SYMPTOMS.—(1) *Pain*: as in biliary colic, but milder (from passage of thick mucus). (2) *Jaundice* usually absent and rarely severe. (3) *Gall-bladder* often palpable (*see* Courvoisier's Law, p. 472).

Attacks may be recurrent. In intervals, nagging pain, ill-health, and gastric disturbance.

DIAGNOSIS of carcinoma not uncommon, and resemblance at operation often close. From gall-stones, very difficult, but no tenderness between 9th rib and umbilicus.

TREATMENT.—Medical treatment as for gall-stones, results often good; operation if symptoms resistant.

Cholecystitis Obliterans (*Atrophic Cholecystitis*).—

ETIOLOGY.—Sequel of gall-stones and chronic cholecystitis.

GALL-BLADDER.—Contracted even to a fibrous cord; may be hugging a stone; adhesions common.

SYMPTOMS.—Pain and ill-health from adhesions; may be passage of some ropy mucus.

Calcification may follow either of above forms.

6. CANCER OF THE GALL-BLADDER AND BILE-DUCTS.

Cancer of the Gall-bladder.

Etiology.—

NATURE OF GROWTH.—*Primary carcinoma.* All others very rare.

AGE.—55 to 65 years.

SEX.—Females 3 or 4 to 1 male.

RELATION TO GALL-STONES.—*Gall-stones present in 75 to 90 per cent.* Note also: (a) Catarrh present in only 10 per cent of secondary growths; (b) Carcinoma develops in 5 to 15 per cent of gall-stones. *Conclusion*: Gall-stones are cause and not result of the carcinoma, another factor (probably chronic inflammation) also being necessary.

Morbid Anatomy.—

CARCINOMA.—Columnar or spheroidal cells. Growth either infiltrates and thickens wall, or projects into lumen as villous fungating mass.

SITE OF ORIGIN.—Fundus usually. Less often, entire bladder, or at neck.

LIVER —Secondary growths in 50 per cent. In others, usually distention by bile
BILE-DUCTS —Frequently involved by spread of growth Original site often uncertain
ABDOMINAL GLANDS —Also affected
Secondary growths rare elsewhere.

Symptoms.—(Elderly woman, often previous history of gall stones)
DISCOMFORT —In right hypochondrium May be PAIN severe and paroxysmal, and superficial tenderness (8th dorsal segment)
JAUNDICE —Occasionally absent
LOSS OF WEIGHT, ANOREXIA
GALL-BLADDER TUMOUR —In over 50 per cent Becomes hard and irregular
LIVER —Usually enlarged
Symptoms are progressive
Jaundice results from liver growths or glands in portal fissure, or bile duct involvement
Supraclavicular gland may be enlarged

Duration.—Six months from jaundice Death in 'cholæmia'.

Complications. —Suppurative cholecystitis, cholangitis Adhesions to pylorus, etc Fistulæ

Diagnosis from Gall-stones.—Age Progressive jaundice and cachexia Palpable gall bladder (*See* Courvoisier's Law, p 472) Secondary growths in liver often decide
Note At operation a chronically inflamed gall bladder may be hard and thick and distinction uncertain
WHEN LIVER INVOLVED Symptoms indistinguishable from hepatic carcinoma and *when bile ducts* involved from carcinoma of bile ducts or head of pancreas

Treatment.—Operation and removal if liver not involved Considerable mortality from hæmorrhage

Other Tumours of Gall-bladder.—*Secondary growths* very rare, no relation to gall stones males preponderate
Innocent tumours, very rare

Cancer of the Bile-ducts.

Etiology.—
NATURE OF THE GROWTH —*Primary carcinoma*
AGE —55 to 65 years
SEX —Males, slight majority
GALL-STONES —Present in about 30 per cent.

Morbid Anatomy.—
CARCINOMA —Usually columnar cells, occasionally spheroidal
GROWTH —(1) Projects into lumen, size not greater than cherry, (2) More commonly, infiltrates wall, producing stricture. *Origin* : commonest near termination. Tends to spread along ducts, even to gall bladder, or into pancreas.

Cancer of the Bile-ducts—Morbid Anatomy, *continued.*

BILE-DUCTS.—Distended above growth.

GALL-BLADDER.—Is always distended, unless prevented by adhesions from previous cholecystitis.

LIVER.—*Deep green colour.* Not always enlarged. Secondary growths in 20 per cent (low percentage ascribed to rapid death from cholæmia).

Symptoms.—*Onset insidious.* Resembles severe catarrhal jaundice with cachexia.

JAUNDICE.—Usually earliest symptom. Steadily *increases to dark green.*

CACHEXIA.—Loss of weight. Anorexia.

PAIN.—Absent or slight: occasionally biliary colic.

GALL-BLADDER.—Palpable: surface *smooth.* Primary growth never palpable.

LIVER.—Usually palpable. Extension of growth may render the symptoms identical with carcinoma of gall-bladder, head of pancreas, or liver.

TUMOUR OF HEPATIC DUCT.—As above, but gall-bladder not enlarged.

TUMOUR OF CYSTIC DUCT.—As cancer of the gall bladder.

Duration.—Six months from onset of jaundice. Death due to 'cholæmia', or suppurative cholangitis.

Complications.—Rare: portal thrombosis, rupture of distended gall-bladder, hæmorrhage from growth.

Diagnosis from Gall-stones.—By (1) Age; (2) Insidious onset, (3) Progressive jaundice and cachexia; (4) Enlarged gall-bladder.

Treatment.—Cholecystenterostomy may temporarily relieve gall bladder and liver.

7. STENOSIS AND OBSTRUCTION OF THE BILE-DUCTS.

Congenital Obliteration of the Bile-ducts.

Situation and Extent of Obliteration.—Varies; usually in common duct, generally extending into common hepatic duct.

Morbid Anatomy.—

BILE-DUCTS.—Great fibrous thickening. Lumen may be recognizable microscopically, but no epithelial cells are present. *No dilatation* above constriction.

LIVER.—Enlarged, hard, and bile-stained. *Histology*: Marked cirrhosis, unilobular, or in parts multilobular.

SPLEEN.—Enlarged.

Pathogenesis.—Origin may be:—

(1) CONGENITAL MALFORMATION.—On this theory, cirrhosis of liver is secondary.

Note.—In adults, neither enlargement of liver nor marked cirrhosis follows obstruction of ducts.

(2) CONGENITAL CIRRHOSIS OF LIVER.—This theory involves inflammation spreading down ducts, an obliterative cholangitis. Supported by size and cirrhosis of liver, and enlarged spleen. (Cirrhosis ascribed to placental toxins.) Probable explanation, but undecided.

(3) CONGENITAL SYPHILIS.—A very rare cause. Constriction by chronic peritonitis is on record.

Symptoms.—*Jaundice*: onset at birth, or within two weeks: progressive and severe. Emaciation and hæmorrhages, especially from cord, usually precede inevitable death, which frequently occurs in convulsions. No pyrexia.

Diagnosis.—From other forms of ICTERUS NEONATORUM (*see* p. 155)

Stenosis of the Bile-ducts.

Stenosis or stricture may be: (1) *Congenital* (*see above*); (2) *Acquired*. Extremely rare as result of gall-stone ulceration, except in cystic duct (*see* CHOLELITHIASIS). Annular carcinoma of duct may simulate stenosis.

Obstruction of the Bile-ducts.

Etiology practically identical with obstructive jaundice.

VI. CHOLELITHIASIS: GALL-STONES.

ORIGIN AND FORMATION OF GALL-STONES.

Gall stones consist mainly of cholesterin and calcium bilirubin. The chief problems are: (1) The origin of the cholesterin; (2) The cause of precipitation; (3) The mode of growth of the stones.

Older Theories.—

(1) STAGNATION AND INSPISSATION OF BILE.—Now known that concentration of bile does not by itself result in precipitation of solids.

(2) THUDICHUM'S CHEMICAL THEORY.—Sodium glycocholate was supposed to decompose, during stagnation, into glycocoll, cholic acid, and a sodium salt; and hence the cholesterin, being little soluble in acid solutions, was deposited, i.e., *the precipitation resulted from a change in reaction*.

These theories accepted presence of cholesterin as due to normal secretion from the blood

Modern Theory: Naunyn's Views.—

(1) ORIGIN OF THE CHOLESTERIN.—(i) Cholesterin and calcium are products of disintegration of cells, i.e., *the result of a mild catarrh of the mucous membrane*. (ii) *Micro-organisms* are the cause of the catarrh.

(2) MODE OF PRECIPITATION.—Calcium combines with bilirubin and forms a precipitate, acting as a nucleus on which cholesterin is deposited.

Gall-stones—Origin and Formation, *continued.*

Naunyn's theory of a 'lithogenic cholecystitis' is accepted fundamentally, especially as to origin of cholesterin, viz., from disintegrating mucous membrane due to action of bacteria.

NOTES ON ACTION OF BACTERIA.—

(i) Bile is favourable medium for bacteria, especially of coli-typhoid group.

(ii) Gall-stones have been experimentally produced by injecting attenuated typhoid cultures into gall-bladders. Gall-stones placed in normal gall-bladders are absorbed.

(iii) Gall-stones are definitely related to enteric fever. Bacilli may be recoverable from stones ten to thirty years after disease. Coli-typhoid bacilli frequently present.

NOTES ON MODE OF PRECIPITATION.—

(i) Cholesterin is also formed in disintegration of mucous membrane other than gall-bladder, and is present in cysts, but does not precipitate. Naunyn's explanation: nucleus of calcium bilirubin absent.

(ii) Pure cholesterin calculi occur. Hence Naunyn's explanation is insufficient.

iii. Cholesterin is soluble in alkaline solutions, especially with bile acids, but little so in acid solutions. On this point, note: (i) Cyst fluids are alkaline; and (ii) Coli-typhoid bacilli are acid producers.

Kramer's theory—*The bacteria, by altering to acid the reaction of the medium, favour deposition of the cholesterin produced by their action on the mucous membrane.*

Accessory Factors (conditions favouring stagnation of bile are admittedly accessory factors)—(1) Sedentary occupations, lax abdominal walls, constipation, 'tight lacing'; (2) Pregnancy; (3) Gastric catarrh.

General Summary.—

1. Cholesterin and calcium result from disintegration of mucous membrane (cholecystitis) due to mild inflammatory action of bacteria.
2. Precipitation depends upon: (a) Presence of a nucleus; (b) Stagnation; (c) Alterations in reaction of medium—probably the essential factor.
3. Growth of a calculus is by deposition and crystallization.

(*See* Adami's *Pathology* for consideration of 'pure cholesterin calculi', and many difficult questions arising. Other unsettled points include: Excretion of cholesterin from the blood by the gall-bladder—revived by Hürtle. Crystallization of cholesterin is complex: certain cholesterin compounds possess in solution the physical properties of solid crystals, i.e., Lehmann's 'liquid crystals'.)

COMPOSITION AND VARIETIES OF GALL-STONES.

1. PURE CHOLESTERIN STONES.—Uncommon. Solitary, large, smooth, yellowish, translucent appearance. Consist of crystalline cholesterin 98 per cent. Usually nucleus of pigment. Formed in gall-bladder when cystic duct blocked.
2. LAMINATED CHOLESTERIN STONES.—Externally resemble pure cholesterin. On section laminæ of cholesterin and calcium biliverdin (green) or calcium bilirubin (brown). Cholesterin forms 75 to 90 per cent.
3. COMMON GALL-STONES.—Mixed cholesterin and calcium bilirubin. Consist of: (i) Nucleus—some débris; (ii) Crystalline body of cholesterin, calcium bilirubin and biliverdin, traces of $CaCO_3$; (iii) Non-crystalline crust. Soft and greasy when fresh, hard when dry.
4. PURE CALCIUM-BILIRUBIN STONES.—Form in hepatic ducts. Size, pea to grain of sand. Shape, irregular. Occur as (i) Soft and brown; (ii) Hard, metallic lustre.
5. Rare forms occur, e.g., calcium carbonate (extremely rare in man, common in animals).

NUMBER.—Often multiple.

SHAPE.—In gall-bladder are roundish. If moderately numerous, are faceted. In common duct, are elongated.

SITUATION.—In gall-bladder only, in over 50 per cent; both in gall-bladder and other sites, in over 30 per cent.

X RAYS.—Calcium is opaque: over age of 40 years, stones often give shadow, but negative result does not exclude calculi.

ETIOLOGY.

AGE.—Usually over 40 years. Rare under 30 years.

SEX.—Females preponderate, 75 per cent. Ascribed to factors causing stagnation of bile.

PREDISPOSING FACTORS (*see* ORIGIN and FORMATION).—Specific infections of gall-bladder, e.g., enteric, and stagnation of bile.

FREQUENCY. In 5 to 12 per cent of autopsies. Commoner in Germany than in Great Britain and America.

DIET.—Influence uncertain. Possibly aided by diet rich in carbohydrate and poor in protein.

SYMPTOMS.

Classification of Symptoms.—Symptoms of gall-stones are very numerous. Arise from: (a) Mechanical effects; (b) Results of inflammation, simple or suppurative, local or general. The symptoms also vary with the site of the stone, the degree of obstruction, and other factors. They are accordingly arranged in this section under the groups: (1) Biliary colic, general account; (2) Obstruction of the cystic duct; (3) Obstruction of the common duct; (4) Remote effects of gall-stones.

LATENT SYMPTOMS.—Symptoms are often latent for long periods, or stones found only at autopsy.

Gall-stones—Symptoms, *continued.*

PRODROMAL SYMPTOMS.—Oppression in epigastrium, relieved by eructations or vomiting: occasional slight rigors: attacks of flatulent dyspepsia: pain in back or right shoulder. Group of symptoms is indefinite, resembles the prodromal symptoms of duodenal ulcer, and rarely leads to diagnosis

1. Biliary Colic.—

GENERAL DESCRIPTION —

PAIN.—

Onset.—Sudden: occasionally previous shivering. Most common at night.

Site.—Right hypochondrium. Not uncommon in epigastrium at onset *Rarely*, left hypochondrium.

Radiates widely—Back, and to right shoulder. Less distinctively, across abdomen.

Character.—Agonizing and paroxysmal. No relief in any position, or from pressure.

Termination—Gradually eases: leaves dull ache. Rarely, sudden relief.

Duration.—Usually three to twelve hours. Subsequently much prostration. May recur after short interval.

Cause of pain—*Muscular spasm* excited by movement of stone in bile passages Accessory causes: (a) Acute inflammation due to stone; also possibly: (b) Shape of Heisterian valves; (c) Distention of gall-bladder by secretion. First attack usually severest: later, ducts dilated.

Accidents during a paroxysm.—Fatal syncope; rupture of gall-bladder. Rare.

RIGOR.—*Sweating.* Anxious expression.

VOMITING.—May ease pain, by relaxing gall-bladder spasm

PULSE.—Small and feeble. Condition of collapse.

TEMPERATURE—Often 102° to 103° (ascribed to inflammation)

TENDERNESS.—Marked at gall-bladder spot—midway between tip of 9th rib and umbilicus. Right rectus often rigid.

LIVER.—Very tender: may be enlarged. Similarly gall-bladder. Palpation during spasm unsatisfactory.

JAUNDICE.—In 50 to 75 per cent. *Onset*: few hours to two or three days after pain commences. Usually transient and slight. Probably mainly from inflammatory swelling blocking duct.

DIAGNOSIS.—Characteristics: (1) Colic; (2) Subsequent jaundice; (3) Often previous attacks; (4) Stone passed in fæces (stir in 1–20 carbolic, and strain through muslin). Diagnosis from:—

RENAL COLIC.—Radiation of pain: no jaundice.

ACUTE GASTRITIS.—Pain less; rigor rare; no collapse.

DUODENAL AND GASTRIC ULCER.—(Closely resembled by chronic gall-bladder with adhesions.) (*a*) Pain daily and regular; (*b*) No radiation to shoulder; (*c*) Less paroxysmal; (*d*) Gastric contents show free HCl increased (in gall-stones normal or usually diminished).

MOVABLE KIDNEY.—Worse by day; pain less; no collapse.

APPENDICITIS.—Biliary colic referred to iliac fossa is very rare.
HYSTERIA.—By other signs. Pain often periodical.
ACUTE CHOLECYSTITIS.—Symptoms may be identical
OTHER CONDITIONS.—Pleurisy and pneumonia at right base. Lead colic. Tabetic crises. Malignant disease. Acute pancreatitis (profound collapse). Acute pyelitis.

2. **Obstruction occurring in Cystic Duct.**—General symptoms of biliary colic. Sequelæ may be :—

(1) DILATATION OF GALL-BLADDER (*hydrops vesicæ felleæ*).—Tumour may be very large. Contents : (*a*) Acute obstruction : bile and mucus (*b*) Chronic obstruction : clear mucus. Sequelæ : (i) Suppuration, specially chronic empyema ; (ii) Atrophy.

(2) ACUTE CATARRHAL CHOLECYSTITIS.—Common, and is largely cause of symptoms

(3) SUPPURATIVE CHOLECYSTITIS.—Either : (i) Acute ; or (ii) *Chronic simple empyema.*

(4) CHRONIC CHOLECYSTITIS AND ATROPHY OF GALL-BLADDER.—Not infrequent. All grades from enlarged hard organ to fibrous cord

JAUNDICE may be absent in cystic duct obstruction, or slight degree from inflammation spreading along duct.

(3) **Obstruction occurring in Common Duct.**—Commonest site is near termination General symptoms of biliary colic.

Three groups : (1) Complete obstruction ; (2) Incomplete obstruction ; (3) Ball-valve obstruction.

1. COMPLETE OBSTRUCTION.—(Rare) Symptoms :—
   JAUNDICE.—Deep ; long duration, *intensity unvarying.*
   GALL-BLADDER.—Not enlarged (unless calculus at junction with cystic duct) Liver enlarged.
   NO SYMPTOMS OF SEPSIS
   BILE-DUCTS.—Dilated : clear fluid Calculus may 'work loose'.
   DIAGNOSIS.—From carcinoma by : (*a*) Biliary colic ; (*b*) Gall-bladder not enlarged
2. INCOMPLETE OBSTRUCTION.—Symptoms :—
   JAUNDICE.—Intensity varying ; long duration.
   GALL-BLADDER.—Not enlarged
   LIVER.—Not enlarged No ascites Spleen may be palpable.
   BILE.—Present in fæces.
   FEVER.—Occasionally : from catarrhal cholangitis.
   MORBID ANATOMY.—Common duct and all ducts dilated : walls thickened, inflamed, but no ulceration. Gall-bladder small : walls thickened adhesions common. Liver often small : fibrosis around ducts.
   COMPLICATION.—*Suppurative cholangitis* (*see* p. 465) : symptoms of intense sepsis, high temperature : rapidly fatal.
3. BALL-VALVE OBSTRUCTION ('HEPATIC INTERMITTENT FEVER').—Special symptoms associated with a movable calculus, usually in ampulla of Vater : 'ague' paroxysms of chills, sweats, and fever.
   JAUNDICE.—Deeper after paroxysm.

Gall-stones—Obstruction in Common Duct, *continued.*

During Paroxysm.—(a) Pain over liver; (b) Vomiting and gastric pain.

Paroxysms.—Of great severity; temperature 103° to 105° may recur daily or periodically, as in malaria.

In Intervals.—Temperature normal. General health remains good. Paroxysms are not proof of sepsis: may recur for years without suppuration.

Morbid Anatomy.—As in Incomplete Obstruction, but pancreatitis also present (*see* p. 485).

Diagnosis.—From: (1) Malaria; (2) Suppurative cholangitis (may be sequel); (3) Carcinoma of bile-ducts or pancreas; (4) Chronic cholangitis without gall-stones.

Treatment.—Removal of gall-stone to prevent suppurative cholangitis or pancreatitis.

4. Remote Effects of Gall-stones.—

1. BILIARY FISTULÆ.—

Intestinal.—(a) Duodenum: most common, calculus may cause intestinal obstruction. (b) Colon: next in frequency: often no symptoms.

Rarely, fistulæ recorded in other directions, gastric, renal, etc.; cutaneous, usually at umbilicus.

2. PERFORATION INTO PERITONEUM.—Usually from acute cholecystitis. Perforation may result in: (a) Local abscess; (b) General peritonitis, or this may follow former. *Symptoms*: Peritonitis with localizing symptoms: (i) Sudden pain near liver; (ii) Rapid jaundice (peritoneal absorption of bile).

3. INTESTINAL OBSTRUCTION (*see* Intestinal Obstruction, p. 439).—Usually elderly women. Stone enters duodenum by fistula, and impacts in ileocæcal valve.

4. ADHESIONS.—Common. Produce vague pains (chronic and nagging) varying with organ affected. Pylorus and stomach common.

5. CHRONIC CHOLECYSTITIS.

6. PANCREATITIS.—Rare. (a) Acute: bile runs up Wirsung's duct, with small ball-valve calculus in ampulla of Vater. (b) Chronic: from extension of inflammation.

7. STRICTURE OF BILE-DUCTS.—*Extremely rare* except in cystic duct. *Symptoms* variable: (i) Cystic duct: colicky pain: previous adhesions prevent enlargement of gall-bladder. (ii) Common duct: progressive jaundice, and great liver enlargement: symptoms identical with carcinoma.

VARIOUS FEATURES.

COURVOISIER'S LAW.—'When the common duct is obstructed by a stone, dilatation of the gall-bladder is rare; when the duct is obstructed by other causes, dilatation is common'. (Great importance in diagnosis.) Absence of enlargement with gall-stones ascribed to previous inflammation causing adhesions and fibrosis,

COROLLARY TO THE LAW.—In jaundice due to gall-stones the gall-bladder is usually small; when due to carcinoma it is usually enlarged.

NOTES ON THE LAW.—(a) Stone in cystic duct: gall-bladder enlarges (jaundice usually absent). (b) Carcinoma with previous stones: gall-bladder small. (c) Chronic cholecystitis: gall-bladder may be enlarged.

SIMPLE ENLARGEMENT OF GALL-BLADDER.—Enlarges directly downwards from tip of 9th rib, or slightly inwards. Superficial, cucumber-shaped. Moves with respiration (unless adhesions). Movable laterally only. (Examine bimanually.)

'GALL-BLADDER SPOT'.—Maximum tenderness is midway between umbilicus and tip of 9th rib.

RIEDEL'S LOBE.—A tongue-like projection from lower edge of liver following previous gall-stones or cholecystitis, and often covering enlarged gall-bladder.

DILATATION OF DUCTS ABOVE OBSTRUCTIONS.—Two forms: (a) Cylindrical; (b) Saccular Rare, but may be enormous, simulating, and diagnosed as, various cysts.

CAMMIDGE'S PANCREATIC REACTION.—Theory is: In active disintegration of tissue of pancreas, a sugar compound is set free, and excreted in the urine, where it is recognized by Cammidge's test. Test said to be positive in 75 per cent of gall-stones in common duct, and negative in 75 per cent of cases of carcinoma of pancreas. In conjunction with this should be considered—

ESTIMATION OF FAT IN FÆCES.—With stones in common duct, fatty acids exceed neutral fat. With carcinoma of pancreas, neutral fat exceeds fatty acids. (*See* p. 384)

TREATMENT.

Medical Treatment.—

GENERAL TREATMENT FOR GALL-STONES AND CHOLECYSTITIS (Bain).—Object is to reduce inflammation of the gall-bladder.

REGULAR LIFE AND EXERCISE; AVOID CHILL AND FATIGUE.

DYSPEPSIA AND DIGESTIVE DISTURBANCES.—Must be corrected. Alkalis. Pancreatic ferment. Olive oil if hyperchlorhydria.

BOWELS.—Daily action. Commence with a few rectal washes. Carlsbad salts or hot aperient water before breakfast. Pill daily:—

℞	Colalin	gr. j	Euonymin	gr. j
	Iridin	gr. j	Leptandrin	gr. ½

DIET.—Ordinary simple mixed diet: moderate quantity: reduce fats and carbohydrates.

FLUIDS.—Give freely: especially mineral waters, e.g., Vichy. If gall-bladder tender, mustard-bran packs for few days. When digestion improves, give hexamine gr. vij in cachets twice daily. If progress satisfactory, continue for three months. Operation if no improvement in three weeks, and if not marked improvement in six weeks.

Gall-stones—Treatment, *continued.*

DURING PAROXYSM.—Inject morph. sulph. gr. ¼ with atropin. sulph. gr. $\frac{1}{100}$. Repeat morphia if necessary. Few inhalations of chloroform until morphia acts.

Drinks of hot water, one pint as hot as possible (sod. salicyl. gr. xx and sod. bicarb. gr. xl may be added—Yeo). Fomentations to epigastrium. (In milder cases may be sufficient, with aspirin; but give morphia unhesitatingly.) After attack, dose of calomel.

Surgical Treatment.—Indications: (1) Recurrent biliary colic. (2) Persistent jaundice. (3) Enlargement of gall-bladder (even without jaundice). (4) Septic symptoms associated with signs of stones: as in empyema of gall-bladder. (5) Certain 'remote' results: adhesions, peritonitis, localized abscess, fistula.

RECURRENCE.—Is extremely rare (if all stones removed).

VII. CIRRHOSES OF THE LIVER.

Conditions characterized by increase of the fibrous tissue of the liver. Various classifications are possible. Clinical types are:—

1. ALCOHOLIC OR PORTAL CIRRHOSIS.—Synonym: Lænnec's cirrhosis. Two types, but clinically practically identical:—
   a. ATROPHIC CIRRHOSIS.—Classically in spirit drinkers.
   b. FATTY CIRRHOSIS. Especially in beer drinkers.
2. HYPERTROPHIC BILIARY CIRRHOSIS, or HANOT'S DISEASE.

   Often included are:—
3. CHRONIC PERIHEPATITIS.
4. SYPHILITIC HEPATITIS.

Various conditions associated with cirrhosis, all very rare: (*a*) Hæmochromatosis; (*b*) Anthracotic cirrhosis, from coal dust; (*c*) Obstructive biliary cirrhosis, from chronic obstruction of extrahepatic bile-ducts. Also Banti's disease.

ALCOHOLIC OR PORTAL CIRRHOSIS.

A chronic degeneration of the liver due to the prolonged ingestion of alcohol, characterized pathologically by increased interlobular fibrous tissue and degeneration of liver cells, and clinically by obstruction to the portal circulation.

Note.—Certain disputed theories are referred to at end of section.

Etiology.—

AGE.—Commonest, 40 to 50 years.

SEX.—Males 2 to 1 female.

ALCOHOL.—Almost invariable antecedent of portal cirrhosis.

Morbid Anatomy.—Two forms: (1) Atrophic cirrhosis; (2) Fatty cirrhotic liver. The essential changes in the liver are: (*a*) Increase of interlobular fibrous tissue; (*b*) Degeneration of liver cells.

1. ATROPHIC CIRRHOSIS OF LIVER.—

   SIZE.—Usually small, sometimes markedly.

SHAPE.—Deformed. Capsule thickened.

SURFACE.—Irregular, with protruding 'hobnails', of size of pea upwards. Firm, cuts with resistance.

ON SECTION.—Pale. Yellowish areas surrounded by translucent strands of fibrous tissue, continuous with depressions on surface, and spreading from portal canals.

HISTOLOGY.—(1) Strands of fibrous tissue, mainly multilobular (enclosing several lobules): but varies, and in places, or when advanced, may be unilobular and intercellular. (2) Liver cells degenerating, especially near periphery of lobule (i.e., in portal spheres), with invasion of fibrous tissue: some fat in cells. Often signs of 'regeneration'.

PORTAL VEIN and main liver branches thickened. Hepatic arteries dilated.

2. FATTY CIRRHOTIC LIVER.—

SIZE.—Enlarged.

SURFACE.—Smooth or slightly granular. Firm, cuts with resistance. Pale, and otherwise resembles 'fatty liver'.

HISTOLOGY.—Fatty degeneration and infiltration of liver cells marked. Other changes as in atrophic form.

OTHER CONDITIONS.—

PERITONEUM.—Usually contains fluid. Surface opaque, often thickened.

STOMACH AND SMALL INTESTINES.—Chronic catarrh due to (*a*) alcohol, (*b*) portal congestion

VEINS OF ŒSOPHAGUS AND GASTRIC CARDIA.—Varicose.

SPLEEN.—Enlarged.

TUBERCULOSIS.—Very common: pulmonary, pleuritic, or peritoneal.

Lungs compressed, if much ascites. Arteriosclerosis, myocarditis, fibrosis of kidneys common (probably alcoholism).

Collateral Circulation.—Principal veins involved:—

(1) ACCESSORY PORTAL SYSTEM OF SAPPEY.—(i) Veins in round ligament connecting at umbilicus with epigastric and mammary veins: may form 'caput medusæ'. (ii) Veins through suspensory ligaments, diaphragm, diaphragmatic veins, and vena azygos to superior vena cava.

(2) ŒSOPHAGEAL AND GASTRIC VEINS.—Large varices at end of œsophagus and cardia.

(3) RETROPERITONEAL VEINS, connecting portal and inferior vena cava branches. Also 'veins of Retzius' forming subperitoneal anastomoses of these systems.

(4) INFERIOR MESENTERIC AND HÆMORRHOIDAL VEINS.—Probably little influence: hæmorrhoids not markedly frequent.

Symptoms.—*May be latent* for years with advanced cirrhosis, if collateral circulation effective: duration longest in fatty cirrhotic liver: otherwise two forms are identical, except greater size and tenderness of liver in fatty type.

Alcoholic or Portal Cirrhosis—Symptoms, *continued.*

Common first complaints are: (1) Dyspepsia; (2) Hæmatemesis; (3) Slight jaundice; (4) Ascites and abdominal swelling.

Symptoms are *obstructive*, due to portal congestion, and *toxic*, due to destruction of liver cells.

OBSTRUCTIVE SYMPTOMS.—

GASTRO-INTESTINAL CATARRH.—(Venous congestion and alcoholism.) (1) Anorexia, nausea and vomiting, especially morning; (2) Tongue furred, breath foul; (3) Constipation, and irregular bowels.

HÆMATEMESIS.—(Œsophageal varices.) Often early, very profuse, recurrent. Severe collapse and fatalities very rare.

OTHER HÆMORRHAGES.—Epistaxis, melæna common.

SLIGHT JAUNDICE.—Often absent, but skin sallow, icteroid tint.

PHYSICAL SIGNS.—

'HEPATIC FACIES'.—Dry, sallow, icteroid skin: conjunctivæ watery: venules on nose and cheeks. Patient thin. Appearance often distinctive in late stages.

LIVER.—Often enlarged and tender: generally palpable, even if small. Hard edge, rough surface. A large fatty cirrhotic liver may diminish rapidly under treatment.

SPLEEN.—Usually palpable.

VENULES AT COSTAL MARGIN.

COLLATERAL CIRCULATION.—*See* p. 475.

ASCITES AND DISTENDED ABDOMEN.—Contrasts with thinness elsewhere. In late stages.

TEMPERATURE.—Fever rarely entirely absent. If marked, suggests tuberculosis.

SECONDARY ANÆMIA.

URINE.—Often reduced. Albumin common.

VARIOUS LATE CONDITIONS.—*Nævi*: spider angiomata, face, neck, back. *Œdema* of feet: general anasarca rare. Various effects of ascites.

TOXIC SYMPTOMS.—Delirium, coma, or a condition of cholæmia or 'icterus gravis' may develop at any time, but usually in late stages.

Complications.—*Ascites. Tuberculosis*, very common: cause of death in 15 to 25 per cent. *Pneumonia. Cholæmia. Chronic nephritis.* Thrombosis of portal vein (rare).

Diagnosis.—In early stages, suggested by alcoholic history, gastritis, and enlarged liver. *Diagnosis definite* with 'hepatic facies', hæmatemesis, physical signs in liver and spleen, and ascites. Difficulties may arise form:—

ENLARGED LIVER IN ABSENCE OF ASCITES.—(1) Passive congestion; (2) Fatty liver; (3) Malaria; (4) Leukæmia and splenic anæmia; (5) Syphilis; (6) Amyloid liver; (7) Biliary cirrhosis.

HÆMATEMESIS.—(1) Gastric and duodenal ulcer; (2) Carcinoma.

ASCITES.—(1) Tuberculous peritonitis; (2) Abdominal neoplasm; (3) Chronic peritonitis; (4) Portal thrombosis (very rare).

Prognosis.—Bad. Death usually 3 years from onset of symptoms: occasionally 8 to 10 years with good collateral circulation. When *ascites* occurs, very bad: this is almost a terminal event.

Treatment.—(Syphilis should be excluded by a Wassermann test.)

EARLY STAGES.—A regular and moderate life. Moderate diet, plentiful fluid, regular bowels (saline aperient); *no alcohol*.

GASTRIC CATARRH.—Bismuth, and treatment as in gastritis.

HÆMATEMESIS.—Inject morphia. Adrenalin (solution 1–1000, ʒss to ʒj by mouth): often ineffectual.

ASCITES.—Treatment mainly to relieve discomfort. Great restriction of fluids inadvisable.

PARACENTESIS.—If volume of fluid affects heart, lungs, or comfort.

OPERATION.—Talma-Morison and modifications. *See* TREATMENT OF ASCITES, p. 505.

Notes on Certain Theories, etc.

PATHOGENESIS OF THE LIVER CHANGES.—A causal toxin arriving in portal blood causes degeneration of liver cells; for a time 'regeneration' occurs. The fibrous tissue increases secondarily, as a 'replacement fibrosis'; later, contracting, it causes atrophy of liver cells, compression of portal branches, and hence 'portal obstruction'. Against this theory is fact that cirrhosis is in excess of liver degeneration at any stage. Probably toxin affects both tissues simultaneously. (For 'Regeneration' and 'Newly-formed Bile-ducts', *see* ACUTE YELLOW ATROPHY, p. 456.)

ACTION OF ALCOHOL.—Theories: (1) Direct poison on liver cells. (Reproduction in animals unsatisfactory.) (2) Produces gastro-intestinal catarrh, whence 'autotoxins' are absorbed. (3) Due to 'higher alcohols' and not to C_2H_5OH. All unproved.

COLLATERAL CIRCULATION.—Good effects: relief of portal circulation. Bad effects: (1) Hemorrhage from [varices]; (2) Much blood escapes 'detoxifying' action of liver.

SPLENIC ENLARGEMENT.—Theories of origin: (1) From congestion: probable cause; supported by rapid enlargement in portal thrombosis; opposed by small spleen in chronic heart lesions (liver acts here as buffer). (2) From toxins causing the cirrhosis, or escaping liver in collateral circulation.

ASCITES.—Origin uncertain. Some authorities consider that *recurrent ascites* (i.e., patient lives to require a second tapping) never results from pure portal cirrhosis, and proves either: (1) Presence of chronic proliferative peritonitis (perihepatitis); or (2) Erroneous diagnosis.

1. HYPERTROPHIC BILIARY CIRRHOSIS.

(*Hanot's Disease.*)

A chronic condition of unknown origin, characterized pathologically by unilobular fibrosis of the liver, and clinically by jaundice, enlarged liver and spleen, and absence of ascites.

Hypertrophic Biliary Cirrhosis, *continued.*

Etiology.—

AGE.—Young adults.

SEX.—Males; very rare in females.

CAUSE.—Unknown; alcohol is not a factor; theory of an infection is based on febrile attacks with leucocytosis.

Morbid Anatomy.—

LIVER.—Enlarged markedly. Heavy. Shape normal. Surface smooth. Colour, *dark green* (late stage). Very hard. On section, surface greenish yellow, strands of fibrous tissue visible. *Histology*: (1) Fibrous tissue increased, *especially unilobular.* (2) Cholangitis. proliferation and desquamation of epithelium in smaller ducts, whence obstruction and blockage with bile thrombi. (3) Numerous 'newly-formed bile-ducts' present (liver 'regeneration'). Degeneration of liver cells not extreme

SPLEEN.—Enlarged: fibrosis and atrophy. Weight: 24 to 36 oz. Gall-bladder, bile-ducts, portal vein and tributaries, normal. No gastro-intestinal catarrh. No ascites.

Symptoms.—Young males. No alcoholic history. Very chronic, four to ten years.

ONSET.—Insidious. *Progressive weakness* and malaise. Abdomen swells.

RECURRENT ATTACKS.—Pain over liver, pyrexia, leucocytosis; often nausea, vomiting, and deeper jaundice. Duration, days to weeks.

JAUNDICE.—Tinge slight at onset, progresses; often finally dark.

LIVER AND SPLEEN.—Greatly enlarged. Edge firm.

Usual symptoms of jaundice, except stools dark. Moderate anæmia. In later stages may be: *skin very dark*; hæmorrhage from gums, etc. (icteric origin)—hæmatemesis rare. *Absent*: ascites, signs of portal obstruction. Dyspepsia slight or absent.

Termination.—Progressive weakness; always fatal. Termination. intercurrent diseases, occasionally 'cholæmia', or icterus gravis during febrile attack.

Treatment.—Symptomatic.

3. CHRONIC PERIHEPATITIS.

(*Sugar-iced Liver. Zuckergussleber.*)

Characteristics.—(1) Capsule enormously thickened; (2) Liver contracted, but little or no interstitial cirrhosis; (3) Varying degrees of chronic perisplenitis; (4) Chronic proliferative peritonitis: and (5) Chronic interstitial nephritis.

CLINICALLY.—(1) Recurrent ascites; (2) No jaundice; (3) Chronic nephritis. All degrees to typical 'chronic proliferative peritonitis', (*see* p. 499)

4. SYPHILITIC HEPATITIS.

See SYPHILIS OF THE LIVER, p. 270.

VIII. ABSCESS OF THE LIVER.

Etiology.—Secondary to conditions outside liver. Two groups :—

(1) **Solitary or Tropical Abscess.**—Due to amœbic dysentery (infection with *Entamœba histolytica*). Rarely more than one.

(2) **Multiple or Pyæmic Abscesses.**—Path of infection :—

(a) SUPPURATIVE PYLEPHLEBITIS.—Through portal vein. Primary focus : appendix ; less commonly, other regions in portal area, especially sepsis of hæmorrhoids and rectum.

(b) SUPPURATIVE CHOLANGITIS.—Through the bile passages Arising from : gall-stones ; very rarely, round worms, hydatids.

(c) GENERAL SEPTICÆMIA OR PYÆMIA.—Through general circulation.

Occasional causes : trauma of liver, hydatid cysts.

In bacillary dysentery and similar intestinal infections, abscess of the liver occurs very rarely, and is not always fatal.

1. TROPICAL ABSCESS. (*Amœbic Abscess.*)

Morbid Anatomy.—*See* AMŒBIC DYSENTERY, p. 92.

Symptoms.—Progress may be : (a) Chronic : symptoms indefinite and slow development, many weeks. (b) Acute : severe symptoms in two to three weeks from onset of dysentery. (c) Subacute, most common : symptoms definite, several weeks from onset.

1. PAIN.—(i) Back and *right shoulder*, (ii) Over liver (perihepatitis).
2. LIVER ENLARGED AND TENDER.—Dullness usually increased upwards in mid-axillary line, from common position of abscess (top of right lobe).
3. ICTEROID TINT.—Rarely deep jaundice. Sometimes absent.
4. CONSTITUTIONAL SYMPTOMS (less marked in chronic forms).—(i) *Fever* : irregular, rising to 103°. (ii) *Rigors*. (iii) *Profuse sweats*. (iv) From septic absorption : muddy complexion, wasting, anorexia, furred tongue.
5. PULMONARY SYMPTOMS AT RIGHT BASE.—Cough and pleurisy. (Inflammation spreading through diaphragm. In later stages, and not invariable.)

Leucocytosis : 10,000 to 25,000 ; mainly polynuclears. May be absent.

No ascites, nor enlargement of spleen.

Perforation.—Occurs into :—

1. LUNGS.—Most common. Either direct into lung or via pleura. Symptoms : (i) Cough ; (ii) Signs at right base ; (iii) 'Anchovy sauce' sputum when lung perforated—contains amœbæ, liver tissue, pus scanty. Prognosis : mortality per cent, recovery often slow (if without emetin treatment).
2. OTHER PARTS.—Externally. Stomach. Peritoneum (local or general infection).

Tropical Abscess of the Liver, *continued.*

Diagnosis.—Difficult in early stages. History of amœbiasis important. Examine stools for cysts, and X-ray chest. Diagnosis from: (1) *Malaria*. Often simulated by recurrent pyrexia and rigors. Protozoa in blood, effect of quinine. (2) Empyema. (3) Gall-stones (hepatic intermittent fever). (4) Pylephlebitis. (5) Suppurating hydatid cysts. Liver puncture when in doubt.

Treatment.—

MEDICAL.—*Emetine* (*see* AMŒBIC DYSENTERY, p. 94): efficacious in many early cases.

SURGICAL.—(1) Evacuation by trocar and cannula (good results); (2) Incision and drainage.

Prognosis.—With emetine and good surgery, mortality rapidly falling. A second abscess may be present and undiscovered.

2. PYÆMIC ABSCESS.

Morbid Anatomy.—

LIVER.—Enlarged, surface smooth, yellow foci of pus often visible under capsule. On section, numerous foci of pus: (1) With suppurative pylephlebitis, foci are in branches of the portal vein. (2) With suppurative cholangitis, foci are in smaller bile-ducts, and gall-bladder and larger ducts are distended with pus.

SINGLE ABSCESS.—Numerous small foci may fuse.

Symptoms.—(1) Severe constitutional symptoms: pyrexia, sweats, rigors. In later stages and extreme forms, often apyrexia and dry skin. (2) Liver enlarged and tender. (3) Icteroid tint. Leucocytosis variable: very high, or often absent. Symptoms of causal condition often mask onset and development.

Diagnosis.—Suspected more easily than definitely diagnosed. Is always secondary to sepsis elsewhere. Invariably fatal. Treatment palliative.

IX. NEW GROWTHS IN THE LIVER.

Secondary malignant tumours are common; all others very rare

MALIGNANT TUMOURS.

Primary Malignant Growths.—Distinction from secondary tumours only by autopsy, after careful search for primary growth (often small, e.g., in rectum). Tend to greater rapidity of growth: jaundice and ascites less common, except type with cirrhosis.

A. CARCINOMA.—Varieties: (1) *'Massive' carcinoma*. Solitary tumour. (2) *Nodular carcinoma*. Multiple growths as in secondary forms. Foregoing are usually spheroidal-celled. (3) *'Carcinoma with cirrhosis'*: probably carcinoma developing in a previously cirrhotic liver, compensatory hyperplasia of the liver cells (excessive 'regeneration') occurring and passing into carcinoma.

B. SARCOMA.—Extremely rare.
Growths corresponding to renal hypernephroma also occur.

Secondary Growths.—*Age*: commonest 40 to 60 years.

A. CARCINOMA.—Common. Liver very large. Nodules on surface, often 'umbilicated': on section grayish or hæmorrhagic: often numerous.

HISTOLOGY.—Character of primary growth, usually columnar-celled. Degenerations common.

SITE OF PRIMARY GROWTH.—(1) Stomach: in 25 per cent. (2) Rectum and colon: also common: often small growth. (3) Other sites in order of frequency: pancreas, bile-passages, uterus, œsophagus, breast, etc.

B. MELANOTIC SARCOMA.—Liver very large. Either: (1) Black nodules; or (2) General infiltration. Metastases present throughout body. Rapidly fatal. Melanuria occasionally.

SITE OF PRIMARY GROWTH. (1) Pigmented mole. (2) In eye (often removed even years previously).

Other sarcomata extremely rare.

Characteristic Symptoms.—

1. LIVER.—*Progressive enlargement*. Discomfort, but often painless.
2. EMACIATION.—Anorexia and gastric troubles common.
3. JAUNDICE.—In 60 per cent., when occurring is permanent and progressive.

Physical Signs.—

LIVER.—(1) Enlarged. (2) Nodular: edge irregular. (3) Nodules often *umbilicated*. Spleen not enlarged.

ASCITES.—In 60 per cent. Moderate: rarely needs tapping.

NODULES AT UMBILICUS AND ALONG LINEA ALBA.—Grow along falciform ligament. Important, not very common.

PYREXIA.—Usually present, about 100°. Spleen not enlarged.

OCCASIONALLY.—Symptoms of primary growth elsewhere in body. Pleurisy at right base, and cough. *Œdema of feet*, late. Superficial abdominal veins dilated (not round navel).

NOTES.—*Jaundice* usually from pressure of lymphatic glands in fissure, or of growth in head of pancreas. *Ascites from pressure* on portal vein, or often from peritonitis. *Enlargement of liver* absent in rare primary nodular type and 'carcinoma with cirrhosis': latter is clinically identical with cirrhotic liver.

Duration.—Three to twelve months.

Diagnosis.—Obvious with characteristic symptoms: (1) Progressively enlarging liver with nodules, often umbilicated; (2) Rapid cachexia; (3) Increasing jaundice, especially if with (4) Ascites. Diagnosis from:—

A. ENLARGED CIRRHOTIC LIVER.—Enlargement not progressive or nodular, emaciation less, history of alcohol. Portal obstruction prominent.

New Growths in the Liver—Malignant Tumours, *continued.*

2. FATTY AND AMYLOID LIVERS.—No jaundice or rapid enlargement, emaciation less. Gummata in amyloid liver may be nodular (Wassermann reaction positive).
3. GALL-STONES IN COMMON BILE-DUCT.—Jaundice and liver diminish from maximum after onset.
4. GUMMATA.—Signs of syphilis, and a positive Wassermann.

Other conditions :—

5. RIEDEL'S LOBE.—Previous gall-stones.
6. HYDATID CYSTS.—Nodules soft, no jaundice or cachexia.
7. HYPERTROPHIC BILIARY CIRRHOSIS.—Rare. Age chronicity, smooth liver, enlarged spleen.

Treatment.—Palliative.

BENIGN TUMOURS.

Rare. Of no clinical importance. *Angioma* or nævus most frequent : size of walnut.

CYSTS.

PARASITIC.—Hydatid.

NON-PARASITIC.—Single or multiple. Occur alone, or with (a) Congenital cystic kidneys ; (b) Other congenital abnormalities.

X. FATTY LIVER.

Two forms : (1) *Fatty infiltration ;* (2) *Fatty degeneration.*

Infiltration is deposit of fat globules in otherwise normal liver-cells. In degeneration, in addition to fat, the cells are degenerated : the fat is said to be mainly deposited from elsewhere in the body. *The two types are often combined,* but typical forms occur.

Causes.—

OBESITY.—Mainly infiltration.

WASTING CONDITIONS.—(1) Phthisis, very common ; (2) Severe anæmia ; (3) Cachexia of any origin.

TOXINS.—(1) Chronic alcohol ('fatty cirrhosis') ; (2) Conditions associated with acidosis, viz., phosphorus (extreme), also delayed chloroform poisoning, diabetic coma, acute yellow atrophy.

DYSENTERY AND DIARRHŒAL CONDITIONS.—Also rarely in enteric.

PHYSIOLOGICAL.—In pregnancy.

Morbid Anatomy.—Liver enlarged : on section pale, often leaves fat on knife. *Histology :* (a) Infiltration : fat mainly in periphery of lobule within normal cells. (b) Degeneration : fat mainly in central zone in granular degenerated cells.

Symptoms.—Indefinite. Those of causal condition. *Liver* enlarged, smooth, and painless. Never jaundice or ascites.

XI. AMYLOID LIVER.

(*Waxy or Lardaceous Liver.*)

Occurs as part of general amyloid disease in young adults with cachexia, usually from chronic suppuration.

Primary Causes.—
1. TUBERCULOSIS.—Especially: (a) Bones, frequent; (b) Lungs.
2. SYPHILIS.—Especially: (a) Bones; (b) Rectum. Suppuration not invariable.

Occasionally rickets, severe fevers, cancer.

Morbid Anatomy.—Liver large, solid, anæmic. On section, glistening surface ('cut bacon'). With iodine, stains dark brown, especially in central zone of lobule. *Histology*: Amyloid material deposited first in subendothelial layer of capillaries in central zone of lobule. Amyloid changes common in other organs: kidneys, spleen, intestines.

Symptoms.—Indefinite. In general amyloid disease: anæmia, wasting, also diarrhœa (if intestines affected). Albuminuria is common. *Liver* enlarged, edge round and smooth. Spleen often palpable.

Diagnosis.—Enlarged liver, with etiological factors present.

Prognosis.—Very bad: progressive emaciation. No cure or treatment.

XII. ABNORMALITIES OF THE LIVER.

Congenital Abnormalities.—(a) Transposition of viscera; (b) Forward tilting (simulates enlargement).

Acquired Abnormalities.—
'LACING' OR 'CORSET' LIVER.—A pressure atrophy followed by fibrosis. Usually a narrow transverse groove of fibrous tissue divides liver into two parts, lower portion reaching almost to umbilicus. Capsule often thickened, and impression of ribs obvious.
'RIEDEL'S LOBE'.—A tongue-like projection following chronic cholecystitis, or gall-stones: often covers an enlarged gall-bladder.
MOVABLE LIVER.—Usually in visceroptosis, in females with pendulous abdomens. Also after recurrent ascites. *See* p. 445.

CHAPTER LXXVIII.

DISEASES OF THE PANCREAS.

I. PANCREATIC INSUFFICIENCY.

Two groups:—

1. **Internal Secretion Deficient.**—*See* DIABETES.
2. **External Secretion or Pancreatic Juice Deficient.**—Voluminous stools result from diminished absorption owing to deficient digestion. Pancreatic juice contains: (i) Trypsinogen, the zymogen of trypsin: protein ferment. (ii) Lipase or steapsin: fat ferment. (iii) Diastase or amylopsin: starch ferment.

Tests of Pancreatic Insufficiency.—
1. INTERNAL SECRETION.—(i) Glycosuria; (ii) Blood-sugar

Diseases of the Pancreas—Insufficiency Tests, ***continued.***

curve and lowered carbohydrate tolerance (*see* DIABETES, p. 327); (iii) Cammidge's test (*see* p. 473).

2. EXTERNAL SECRETION.—Numerous tests exist: negative result in one or even all does not exclude pancreatic disease (*see* Garrod and co-workers, *Lancet*, 1920). (i) Steatorrhœa; (ii) Azotorrhœa; (iii) Diastase estimation in fæces and urine. Also: Sajodin test, Sahli's test, and others (of little value).
3. INTERFERENCE WITH THE SYMPATHETIC NERVOUS SYSTEM.—(i) Signs of hyperthyroidism; (ii) Loewi's adrenalin mydriasis reaction.

'**Pancreatic Stools**'.—Result from deficiency of ferments. Characteristics are: (1) Bulky; (2) Frothy; (3) Oily; (4) Light colour, mainly due to fat. Chemical alterations in the stools are:—

STEATORRHŒA.—Excess and abnormality of fat. *See below*

AZOTORRHŒA.—Of protein in food, amount recoverable in fæces is normally about 5 per cent, but in pancreatic disease 30 to 40 per cent. Normal variations are considerable, and results are greatly influenced by: (*a*) Diarrhœa, which increases amount; (*b*) Constipation, which decreases amount owing to putrefaction. All tests to measure action of trypsin are complicated by the same factors, and are of little value

Fat in Fæces.—Two factors influence this: (1) *Pancreatic secretion*, i.e., lipase. This splits neutral fat of food into glycerin and fatty acids, the latter partly combining with alkalis to form 'soaps'. The fatty acids and soaps (i.e., the 'split fat') can be absorbed, but not the neutral fat. (2) *Bile*. This aids absorption of 'split fat', but has no part in its formation from neutral fat. Hence: (1) *If pancreatic secretion be deficient*, results are (*a*) large amount of undigested fat, (*b*) abnormal relative percentage of neutral fat. (2) *If bile be deficient*, results are (*a*) large amount of undigested fat, (*b*) abnormal relative percentage of 'split fat.'

NOTE.—In diarrhœa, *fat-splitting bacilli* in intestine are numerous, and intestinal absorption is also reduced, result being a high amount of 'split fat'.

FAT EXCRETION AND PANCREATIC REACTION.

Type of Case	*Total Fat* (per cent of dried fæces)	*Form of Fat in Excess*	*Cammidge's Pancreatic Reaction*
Normal	20 to 40	Equal	Negative
Carcinoma of pancreas ..	70 to 80	Neutral	Negative in 75 per cent
Chronic pancreatitis ..	50 to 80	Neutral	Usually positive
Calculi in common duct..	60 to 70	Split	Positive in 75 per cent
Carcinoma of common duct	60 to 70	Split	——

II. PATHOLOGY OF ACUTE PANCREATIC LESIONS.

The Origin of Lesions of the Pancreas.—Destruction of the pancreatic tissue by the trypsin of its own secretion (i.e., autolysis) is probably the basis of many pancreatic lesions.

Pathogenesis of *'hæmorrhagic necrosis of the pancreas'*. Probable sequence:—

1. Pancreatic secretion infiltrates tissues of pancreas, owing to obstruction of its exit.
2. Necrosis of the pancreatic cells and *blood-vessels* results. This autolysis is due to trypsin, and is not 'fat necrosis'.
3. Hæmorrhage thus occurs into, and then extends beyond, the gland, with consequent escape of secretion, and 'fat necrosis' of surrounding tissues (*see below*).

OBSTRUCTION TO THE PANCREATIC SECRETION.—May arise from: (1) *Action of gall-stones*, usual cause. (2) *Gastric juice and duodenal contents* entering the gland. This possibly occurs from injury to duodenal papilla by: (*a*) Gall-stones; (*b*) Vomiting and gastritis. Other causes may be: (3) Cancer. (4) Trauma. (5) Pancreatic calculi. (6) Parasites entering duct. (7) Alcohol, viz., co-existing cirrhosis of liver and pancreas.

[The pancreas has two ducts: (1) *Wirsung's* main duct, joins the common bile-duct in ampulla of Vater; (2) *Santorini's* accessory duct, independent opening into duodenum. Note: (i) In 90 per cent the two ducts connect and hence a partial alternative path exists; (ii) In 10 per cent of all cases Santorini's is the main duct.]

ACTION OF GALL-STONES ON THE PANCREAS.—Methods by which gall-stones affect the pancreas.

1. *Stone in the ampulla of Vater.*—
   i. A small stone may obstruct the exit without occluding either the common bile or Wirsung's duct. Hence, *bile passes up Wirsung's duct*, and together with the pancreatic secretion causes *hæmorrhagic necrosis* and its sequelæ. Undoubted origin of most acute pancreatic lesions. Rarity is due to: (*a*) Stone in ampulla blocks one or both ducts in at least 70 per cent; (*b*) Anatomy of ducts.
   ii. When Wirsung's duct is blocked, *chronic pancreatitis* results from retention of secretion.
2. *Passage of a stone may enlarge duodenal papilla.*—Thus *duodenal contents* can enter Wirsung's duct, *causing hæmorrhagic necrosis* and sequelæ.

   Note: Duodenal contents cannot enter normal papilla owing to valve action. Both bile and gastric juice (or duodenal contents) experimentally injected into Wirsung's duct produce lesions identical with hæmorrhagic necrosis and its sequelæ.

Pathology of Acute Pancreatic Lesions, *continued.*

(3) *Inflammation spreads to pancreas from common bile-duct,* due to passage of a calculus, causing *chronic pancreatitis* (this swelling may itself compress duct later, and prolong jaundice)

Fat Necrosis.—

MODE OF PRODUCTION (*see above*) —Pancreatic juice, liberated by necrosis and hæmorrhage meets fat of its own and other tissues near, and by its lipase (fat splitting ferment) produces *fat necrosis* Thus presence of fat necrosis is *proof of hæmorrhagic necrosis of pancreas*

CHEMICAL CHANGE —The fat is split into glycerin which is absorbed, and fatty acids which are deposited as *opaque needle like crystals* often combined with calcium

PATHOLOGICAL CHARACTERS —*Sites* Interlobular pancreatic tissue mesentery, omentum, and abdominal fat especially near pancreas *Appearance of foci* (*a*) Size of pin's head (may be larger), (*b*) Opaque white (*c*) Sharply defined suggestive of miliary tubercles but not raised May appear within three hours of lesion *Pancreas* Lobules separated by dead white areas (*see* ACUTE PANCREATITIS)

III. ACUTE PANCREATITIS.

(*Hæmorrhagic Necrosis of the Pancreas*)

'Hæmorrhagic necrosis of the pancreas' (Opie) is the more correct term, since steps are (1) Tryptic necrosis (2) Hæmorrhage (3) Inflammation, which is absent at onset

Clinical Groups.—Various conditions usually known as follows are recognizable, but—of varying severity are of similar etiology (1) *Pancreatic hæmorrhage or apoplexy* fatal in a few hours rare (2) *Acute hæmorrhagic pancreatitis,* fatal in two to five days occasional recovery (3) *Gangrenous pancreatitis* subacute fatal in weeks or months (4) *Acute suppurative pancreatitis* occurs when bacteria are present Certain *pancreatic and peripancreatic cysts* are sequelæ of the acute lesions

1. PANCREATIC HÆMORRHAGE.

Of medico-legal importance as a cause of rapid death in those apparently in good health

Pathologically to be regarded as a severe form of hæmorrhagic necrosis of the pancreas.

2. ACUTE HÆMORRHAGIC NECROSIS.

Usually in adult males

Cause.—Obstruction to pancreatic secretion (*see* PATHOLOGY OF ACUTE PANCREATIC LESIONS) or trauma.

Morbid Anatomy.—

PANCREAS.—Swollen. On section, mottled appearance: infiltration with altered blood. *Histology*: Necrosis of parenchymatous cells of blood-vessels and interstitial tissue, much blood; inflammatory changes at margin of necrosis.

HÆMORRHAGE.—In tissues of and around pancreas: often in lesser sac.

FAT NECROSIS.—*See above.*

GALL-STONE.—May be present in gall-bladder or ampulla of Vater.

Symptoms.—Onset often preceded by dyspepsia and gastric pain. Previous biliary colic not infrequent. Chief features are:—

SUDDEN ONSET.

PAIN.—Severe and paroxysmal in upper abdomen.

SHOCK AND COLLAPSE.—Small rapid pulse. Cold skin.

VOMITING.—Early, copious, and bile-stained. Rarely fæcal.

ABDOMEN DISTENDED.—Above umbilicus. Tumour absent or appears late. Constipation.

TEMPERATURE.—Low at onset: may rise, or remain subnormal.

Leucocytosis usual. Jaundice occasionally. Glycosuria very rare.

Death on second to fourth day, or earlier ('pancreatic apoplexy'). Occasional recovery.

Diagnosis.—Difficult. Especially from: (1) *Peritonitis*, e.g., perforated peptic ulcer; (2) *Acute intestinal obstruction*; (3) *Gall-stones.*

Treatment.—Laparotomy. Search for gall-stone, and remove if present. Otherwise operation as rapid as possible.

3. GANGRENOUS PANCREATITIS.

Etiology.—A later stage of last condition, hence develops in forms with *less acute onset*. Very rare. In acute hæmorrhagic pancreatitis surviving one week, pancreas is found dry and reddish-black; after (about) two weeks is black and friable, later, an offensive black fluid in lesser sac, condition constituting 'gangrenous pancreatitis'. General peritonitis rare owing to adhesions.

Symptoms.—As in last condition, but diminishing after fourth day. Subsequently: (1) *Fever and signs of sepsis.* (2) *Tumour* above umbilicus, stomach above and colon below, extending towards spleen, due to fluid in lesser sac. Other symptoms may be: Epigastric pain and tenderness, vomiting, leucocytosis, diarrhœa common, jaundice occasionally. Glycosuria very rare.

Treatment.—Evacuate fluid. Recovery extremely rare.

4. SUPPURATIVE PANCREATITIS.

(*Abscess of Pancreas.*)

Etiology.—(1) Often no cause found. (2) Gall-stones present: suppurative cholangitis may exist, whence suppuration spreads along Wirsung's duct. Possibly: (3) Duodenal contents enter Wirsung's duct owing to injury of papilla either by gall-stones or by catarrh and vomiting. Acute hæmorrhagic pancreatitis may precede abscess.

Abscess may be single or multiple.

Suppurative Pancreatitis, *continued.*

Symptoms.—Usually indefinite. Often previous attacks of pain and vomiting. Principal symptoms: (a) Fever and sepsis; (b) Epigastric tumour (often absent). Rare are jaundice and glycosuria.

Sequelæ.—May be: (*a*) Peripancreatic abscess; (*b*) Perforation of abscess into stomach, duodenum, or peritoneal cavity; (*c*) Thrombosis of portal vein.

Treatment.—Operation. Abscess, and usually fat necrosis, present.

5. SUBACUTE PANCREATITIS.

A form occurs in mumps, characterized by pain in upper abdomen: prognosis always good. Slight injuries or hæmorrhage of pancreas possibly produce similar attacks of pain over prolonged periods.

IV. CHRONIC PANCREATITIS.

Chronic interstitial pancreatitis occurs in two histological types, which differ also in clinical manifestations, and probably in etiology.

1. Chronic Interlobular Pancreatitis.—

ETIOLOGY.—Cause arises from ducts.

1. Partial or complete occlusion of Wirsung's duct (usual origin) from: (*a*) Gall-stones in ampulla; (*b*) Carcinoma, (*c*) Pancreatic calculi
2. Inflammation of bile-ducts, due to gall-stones spreads to surrounding pancreas (Pancreas normally surrounds bile-duct in 60 per cent.)
3. Inflammation ascends duct from gut.

MORBID ANATOMY.—Pancreas hard *Histology*: Strands of fibrous tissue between lobules; in early stages, cells of lobules little affected, but later degenerate; *islands of Langerhans persist until fibrosis extreme.* Pancreatic calculi may be present

SYMPTOMS.—Very indefinite. May be either: (1) As in cholelithiasis; (2) Vomiting and gastro-intestinal; (3) Due to, and, as in, carcinoma; (4) Glycosuria—very rare.

2. Chronic Interacinar Pancreatitis.—

ETIOLOGY.—Doubtful. Possibly toxin from blood-stream.

MORBID ANATOMY.—Little macroscopic change. *Histology*. Diffuse fibrosis invading acini; *islands of Langerhans degenerate early*; distribution irregular; interlobular fibrosis slight.

SYMPTOMS.—*Glycosuria* occurs early. (This form of pancreatitis is the predominant pancreatic lesion of diabetes, similar change also in hæmochromatosis.)

V. PANCREATIC CYSTS.

Decision whether origin of a cyst is from pancreas or other structure is often difficult (*see* MESENTERIC CYSTS, p. 502).

Morbid Anatomy.—

RETENTION CYSTS.—*True cysts.* Due to obstruction of main

ducts: also from chronic interstitial pancreatitis blocking small ducts, or sequel of acute pancreatitis. Rarely large.

PROLIFERATION OF EPITHELIUM AND CYSTO-ADENOMA. - Multilocular. Very rare.

PSEUDOCYSTS.—Hæmorrhage and fluid in lesser sac. Etiology: trauma, acute hæmorrhagic pancreatitis.

HYDATID CYSTS.—Extremely rare.

Symptoms.—

TUMOUR.—Round tumour above umbilicus, median or slightly to left. Smooth, spherical, fluctuates, often movable, rarely moves with respiration. *Relation to neighbouring organs* may be: (1) Stomach above and colon below, most common (well exhibited by inflating colon), (2) More rarely, appears above or behind stomach, and occasionally below colon. Often no symptoms until very large. May exist for years.

PAIN.—In epigastrium or back.

NAUSEA AND VOMITING.—Occasionally, from pressure on stomach.

Rarely: jaundice (cyst in head of pancreas); glycosuria; pancreatic stools.

Contents of Cyst.—Reddish fluid. Alkaline. Contains blood and cholesterin. Also contains ferments: most important is proteolytic ferment, since fat and starch-splitting ferments occur in other exudates, but proteolytic ferment may be absent, owing to antitryptic action of blood, also it is occasionally present elsewhere.

Diagnosis.—Chief features are character of cyst, and situation and relation to other organs. Diagnosis from mesenteric and retroperitoneal cysts usually impossible. From hydatid cysts, *hydronephrosis*, and *ovarian cyst*, by above features.

Treatment.—Preferably by partial removal and drainage: obstinate pancreatic fistula, and digestion at edges of wound, often troublesome. Total removal rarely possible. Aspiration dangerous, and reaccumulation usual.

VI. TUMOURS OF THE PANCREAS.

Varieties.—*Carcinoma* is the common tumour, usually of head. Sarcoma, adenoma very rare. Pancreas is frequently invaded by growths of stomach, bile-ducts, etc., and site of primary growth often uncertain.

Symptoms.—

EPIGASTRIC PAIN.—Often severe paroxysms. (Possibly from cœliac ganglion.)

JAUNDICE.—Intense, permanent, and progressive.

GALL-BLADDER ENLARGED.—Not always palpable.

RAPID EMACIATION.

NAUSEA AND VOMITING common.

Very rare are: epigastric tumour; glycosuria; fatty stools.

Tumours of the Pancreas, *continued.*

Diagnosis.—*From gall-stones* by: (1) Rapid emaciation; (2) Gall-bladder usually enlarged (Courvoisier's Law); (3) Jaundice appears gradually, is progressive, and does not intermit. *From carcinoma of bile-ducts, duodenum, stomach, or liver* when compressing the common bile-duct, is usually indistinguishable.

Treatment.—Palliative.

VII. PANCREATIC CALCULI.

Etiology.—Unknown. Are formed in the ducts. No relation to carcinoma.

Characters. — Small. Nearly always multiple. Opaque white. *Composition*: inorganic salts, viz., calcium carbonate or phosphate. *Opaque to X rays.*

Morbid Anatomy. — Behind stone ducts are dilated, and chronic interstitial pancreatitis is usually extreme. Rarely suppuration and abscess formation occur.

Symptoms.—Indefinite Colicky epigastric pain, and vomiting. Occasionally transient jaundice, glycosuria, fatty stools.

CHAPTER LXXIX.

DISEASES OF THE PERITONEUM.

I. ACUTE GENERAL PERITONITIS.

Etiology.—May be primary or secondary.

1. PRIMARY PERITONITIS.—(i) *Idiopathic*: following cold or exposure: no other evident cause. Rare. Usually pneumococcal. (ii) *Terminal*: in chronic nephritis, arteriosclerosis, etc.
2. SECONDARY PERITONITIS.—Due to: (i) *Perforation* usual origin, especially of appendix, stomach, and duodenum, in enteric and dysentery. (ii) *Extension of inflammation*: from cancer, acute inflammation of neighbouring organs, stomach, intestines, pelvic viscera (e.g., puerperal peritonitis). (iii) *Infection by the blood-stream* in septicæmia and pyæmia.

Morbid Anatomy.—

INTESTINAL COILS.—Distended, owing to paralysis and accumulation of gas: adherent through lymph and exudation in various degrees.

PERITONEUM.—Red, injected, and early loss of lustre. Exudation forms.

EXUDATION.—Amount and character varies: (1) Fibrinous, much lymph with little serum. (2) Sero-fibrinous, much serous fluid, lymph on coils. (3) Purulent: pus may be thin, or opaque and

creamy. Occasionally: (4) No exudation, but peritoneum widely injected; severe type, usually streptococcal and puerperal. (5) Gas present (gas-forming anaerobes) in perforation of viscus. (6) Hæmorrhagic, especially cancer.

Bacteriology.—

Most frequently: (1) *B. coli communis* and bacilli of colon group in numerous varieties, including *B. pyocyaneus*, Friedländer's bacillus; (2) Streptococcus, often associated with *B. coli*; (3) Pneumococcus; (4) Staphylococcus. Other bacteria may be: Anaerobic bacilli. Gonococcus. Enteric group. Very rarely: *B. influenzæ* and others.

Special Types.—

PNEUMOCOCCAL PERITONITIS.—Usually in children, 75 per cent being female. Often 'idiopathic', without obvious cause, possibly from genital organs: occasionally otitis media, pneumonia, and other pneumococcal infections. Two varieties:—

1. DIFFUSE.—Acute onset, pain, pyrexia, vomiting, diarrhœa: rapid death. Severe toxæmia may obscure abdominal signs.
2. CIRCUMSCRIBED.—Resembles appendicitis: abscess forms. Diarrhœa and wasting usual. Less fatal.

Pus characteristic, thick, yellow, odourless, with large flakes of fibrin.

GONOCOCCAL PERITONITIS.—In females, usually by extension from gonorrhœal salpingitis. May be diffuse. Usually pelvic. Pain and rigidity of lower abdomen, with gonorrhœal discharge. In males, extremely rare

TREATMENT.—Rest. Vaginal douches Abdominal fomentations. Laparotomy if constitutional symptoms increasing.

PUERPERAL PERITONITIS.—Following parturition, commonly second to fifth day, especially primiparæ Usually streptococ marked by septicæmic symptoms offensive uterine discharge. Spreads through uterus or Fallopian tubes, pain commencing in lower abdomen: great distention Fatal about sixth day.

LATENT TYPE.—In old persons, e.g., in Bright's disease. symptoms slight. In enteric fever: symptoms may be slight, from dull mentality: suggested by falling temperature and rising pulse.

Symptoms.—

ONSET.—(1) *Abdominal pain*: intense, often sudden, increased by pressure, and by all movements; at complete rest may be slight; widespread, or referred to umbilicus. (2) *Abdominal tenderness*: often extreme. (3) *Abdominal rigidity*. (4) Vomiting. (5) *Decubitus*: lies on back, knees drawn up, shoulders raised, arms above head. *Respiration* shallow and costal. *Temperature usually subnormal*. In septic cases, chills and rigors.

'PERITONISM'.—Term applied to group of symptoms, abdominal pain, vomiting, and shock, common to sudden involvement of peritoneum from any cause, e.g., rupture of any viscus.

'LATENT PERIOD'.—For short period, initial symptoms may improve, and almost subside, before those of peritonitis develop.

Acute General Peritonitis, *continued.*

PROGRESS.—

FACIAL ASPECT.—Important sign of the 'acute abdomen': anxious expression, pinched and pallid, eyes sunken. Develops into '*facies Hippocratica*': eyes sunken, nose sharp, temples and cheeks collapsed, face livid, drawn, and anxious.

ABDOMEN.—(1) Distended and tympanitic (from intestinal paralysis); may be fluid and sometimes gas. (2) Immobility, no respiratory movement. (3) Tenderness extreme. (4) Muscular rigidity ('board-like').

VOMITING.—Early symptom; small amounts; painful, but with little effort or retching. First, stomach contents; then bilious; finally thin, slightly fæcal fluid.

CONSTIPATION.—May be motion at onset: but subsequently constipation is complete for both fæces and flatus. Diarrhœa occurs in puerperal and sometimes in pneumococcal forms

PULSE.—Rapid (110 to 150), small volume, high tension or 'wiry'; later, as the heart fails, becomes low tension or 'thready'.

TEMPERATURE.—Usually rises, often to 104°. May fall with later progress.

TONGUE.—Early moist fur. Later: dry and brown.

URINE.—Either frequency or retention.

BLOOD CHANGES.—Marked leucocytosis (20,000 per c.cm. and upwards), with relative increase of polynuclear neutrophils (75 to 90 per cent). (*See also* LEUCOCYTOSIS and LEUCOPENIA.)

NOTES ON SYMPTOMS.—

TEMPERATURE.—Subnormal on occurrence of perforation; then rises, but falls as symptoms progress; in severe cases often no rise. Hence unreliable sign.

TENDERNESS.—To light pressure. Area corresponds to peritoneum, and is usually absent on palpation in loin posteriorly.

ABDOMINAL SIGNS.—(1) *Liver dullness* often greatly diminished in mammary line, but always present in axilla. (2) *Fluid* generally present, but recognition is usually difficult; may be movable dullness in flanks. (3) *Gas* may escape from a viscus.

Rarely, abdomen flat and rigid throughout course.

ABDOMINAL AUSCULTATION.—Complete silence on listening for long periods.

Termination and Prognosis.—Death in two to seven days, with few exceptions, in absence of operation. Pulse becomes feeble and irregular, skin and extremities cold, general lividity, and collapse.

PROGNOSIS.—In event of operation, depends mainly on (*a*) pulse, (*b*) facies.

BACTERIOLOGICAL TYPES.—*Streptococcus*: all fatal. *Pneumococcus*: diffuse type is fatal, localized type has good prognosis. *Gonococcus*: mortality low. *B. coli*: mortality depends largely on early operation.

Diagnosis.—*Characteristic symptoms* (1) Abdomen pain, tenderness, distention, rigidity, and, later, effusion (2) Vomiting and constipation (3) Rapid pulse (4) Facial aspect (5) Shock and collapse Leucocytosis high

DIAGNOSIS FROM OTHER CONDITIONS —

1 INTESTINAL COLIC —Constipation, lead, etc Also renal colic Differ in intermittent paroxysms, pain eased or not increased by pressure

2 ACUTE ENTERITIS —Differs in diarrhœa, pain colicky

3 ACUTE INTESTINAL OBSTRUCTION —Early stages abdomen not distended or rigid (with exceptions, e g, volvulus) vomiting profuse and fæcal pain colicky Early diagnosis may be impossible later peritonitis may co exist

4 INTERNAL HÆMORRHAGE — Especially with ruptured tubal pregnancy and enteric Extreme pallor, and breathlessness

Rarely —

5 HYSTERICAL PERITONITIS — Simulation may be complete

6 ACUTE HÆMORRHAGIC PANCREATITIS — Distention and collapse extreme Vomiting copious

Occasionally —

7 ACUTE PNEUMONIA —Note facies and pulse respiration ratio May be abdominal pain and vomiting

8 TWISTED OVARIAN CYST Tumour present

9 TORSION OF TESTIS One undescended

DIAGNOSIS OF ORIGIN —Previous illnesses may be guide *In perforation of gastric and duodenal ulcer* Generally previous dyspepsia *Appendicitis* Commonest cause with previous good health, especially in children *Enteric fever* Sudden pain and tenderness, rapid pulse falling temperature

Treatment.—

OPERATION —Except in gonococcal type

While diagnosis is doubtful, give no drugs or food fomen on may ease pain (avoid turpentine, to save skin) Flui as desired *After diagnosis* morphia permissible while awaiting operation but never while in doubt

II. INTRAPERITONEAL ABSCESS: SUBPHRENIC ABSCESS.

Principal types (1) Appendix abscess (2) Pelvic abscess (3) Subphrenic abscess Also closely similar (4) Acute diverticulitis

Suppuration may spread to or arise in various areas on the abdominal surface of the diaphragm, constituting the difficult group known as subphrenic abscess' *

SUBPHRENIC ABSCESS.

Anatomical Relations and Varieties of Abscess.—Peritoneal reflections on the superior and posterior hepatic surfaces divide this area into (4) Right and left, by the falciform ligament,

* See Barnard's *Contributions to Abdominal Surgery*

Subphrenic Abscess—Anatomical Relations and Varieties, *continued.*

(c) Anterior and posterior, by coronary and lateral ligaments. Spread of intraperitoneal suppuration is thus partially limited, giving rise to the following varieties of abscess :—

1. RIGHT ANTERIOR (INTRAPERITONEAL) POUCH.—
   RELATIONS.—On left, falciform ligament. Above, diaphragm. Below, liver. Posteriorly, right lateral ligament. In front, adhesions between transverse colon, diaphragm, and lower edge of liver. In absence of adhesions, is continuous with right posterior pouch, round right edge of right lateral ligament.
   ORIGIN OF ABSCESS.—Appendix. Perforation of duodenal and gastric ulcers. Rarely, liver abscess.
2. LEFT ANTERIOR (INTRAPERITONEAL) POUCH.—Also known as *perigastric*, or perisplenic pouch.
   RELATIONS.—To right, falciform ligament. To left, spleen. Below, liver and stomach. Above, diaphragm. Behind, left lateral ligament
   ORIGIN OF ABSCESS.—Perforation of gastric ulcer.
3. RIGHT POSTERIOR (INTRAPERITONEAL) POUCH.—Also known as *subhepatic* or right kidney pouch.
   RELATIONS.—Complex. Below, right kidney and transverse colon. Extends upwards to right and left between liver and diaphragm, with the folds of the coronary ligament between.
   ORIGIN OF ABSCESS.—Appendix. Occasionally stomach and duodenum.
4. LEFT POSTERIOR (INTRAPERITONEAL) POUCH.—Formed by *lesser sac* of peritoneum. Foramen of Winslow closed by adhesions.
   ORIGIN OF ABSCESS.—Perforation of gastric ulcer.
5. EXTRAPERITONEAL.—On 'bare area' of liver.
   ORIGIN OF ABSCESS.—Liver abscess, or ruptured hydatid cyst.

MOST FREQUENT TYPES.—Right and left anterior. In perforation of peptic ulcer, its position right or left of falciform ligament influences the direction of spread.

Limitation to pouches described is not absolute, and two, or parts of two, may be involved.

Commonest Causes.—(1) Perforation of gastric or duodenal ulcer; (2) Appendicitis, before or after operation.

Symptoms.—

DUE TO PERFORATED ULCERS.—Initial symptoms of perforation. These subside as localization occurs. After about ten days, symptoms of suppuration develop: *pyrexia* (rarely exceeds 102°), wasting, rigors or chills, irregular constipation or diarrhœa, with some pain in upper abdomen and increased respiration.

ARISING FROM APPENDICITIS.—Onset often insidious, with gradual development of symptoms of suppuration.

Physical Signs.—Vary with (1) Presence or absence of gas, in absence of gas, may simulate empyema (2) Position of abscess

GAS PRESENT —(1) A small amount often escapes on perforation appears as movable bubble (diameter about 1 inch) in anterior varieties resonant area either in epigastrium or behind ribs according to patient's position This movable 'bubble of air' is of great diagnostic importance, but entails careful examination (2) Large amounts may escape from viscus, or form subsequently (anaerobic bacteria) physical signs closely simulate pneumothorax *pyopneumothorax sub phrenicus Very rare*

GAS ABSENT —

1 RIGHT ANTERIOR POUCH —(i) *Abdominal signs* Epigastrium rigid Palpable mass from under costal margin, dull on percussion being limited on left by falciform ligament does not extend beyond mid line, but outline to left curved from bulging of ligament From presence of adhesions dullness does not move on respiration, and does not extend downwards beyond normal hepatic limits (ii) *Thoracic signs* Diaphragm may be pushed up, with dullness and deficient breath-sounds at base of lung Heart may be displaced up but not laterally

2 LEFT ANTERIOR POUCH —Similar to above, but on left of falciform ligament

3 RIGHT POSTERIOR POUCH (subhepatic) —Signs difficult No swelling Tenderness and rigidity in right loin Dullness and deficiency of breath-sounds at right base, heart not displaced

4 LESSER SAC —Tumour dull on percussion presenting below or occasionally above stomach often absent (Pancreatic pseudocysts) Diagnosis mainly by symptoms

5 EXTRAPERITONEAL — Diaphragm displaced up and down Moves on respiration Signs at right base

Course.—

WITHOUT OPERATION —(1) May perforate diaphragm extraperitoneal type sometimes into pleura other forms progress more slowly, hence pleural adhesions form, and rupture occurs into lung, with severe cough and expectoration Occasionally discharge into intestines (2) Chronic sepsis, fatal Mortality without operation, 75 per cent

WITH OPERATION AND EFFICIENT DRAINAGE —Mortality at least 30 per cent.

Diagnosis.—Important are —

HISTORY —Previous peptic ulcer, and symptoms of perforation, appendicitis, abdominal operations Interval after acute symptoms, few days to several weeks *often ten to twenty days*

SYMPTOMS OF SEPSIS —Temperature rarely exceeds 102°

PHYSICAL SIGNS —Often both abdominal and thoracic (base of right lung from extension of inflammation through diaphragm). Note 'bubble of air'.

Subphrenic Abscess—Diagnosis, *continued.*

X RAYS.—Displacement of organs, and abnormal shadows.

NEEDLING.—In lower intercostal spaces over dullness: along vertebral border of scapula. Test for pus to depth of three inches. Needle must always be completely withdrawn before inserting in a different direction. Many punctures often necessary.

DIAGNOSIS FROM.—Principally:—

1. EMPYEMA.—In absence of gas. Pleural effusions and changes in the lung may co-exist with subphrenic abscess.
2. TROPICAL ABSCESS OF LIVER.
3. PERINEPHRIC ABSCESS.

Rarely:—

4. PANCREATIC DISEASE.—In lesser-sac abscess
5. PNEUMOTHORAX.—With large amounts of gas (very rare).

Treatment.—*Operation.*

PELVIC ABSCESS.

Secondary to inflammation of Fallopian tubes, around uterus, or appendix. Symptoms of sepsis, with tenderness of lower abdomen, on examination per rectum or per vaginam, tender swelling, often fluctuating.

DIVERTICULITIS. (*See* p. 197.)

III. CHRONIC PERITONITIS.

Varieties.—

1. TUBERCULOUS PERITONITIS.—*See* pp 160, 164
2. CANCEROUS PERITONITIS.—*See* p. 502.
   Not further referred to in this section. In some instances resembles the conditions found in the types following.
3. CHRONIC ADHESIVE PERITONITIS.—*Extension of inflammation from underlying structures.*
   a. Local: Especially (i) Pelvic; (ii) Liver and spleen, (iii) Diverticulitis, pericolitis, and intestinal adhesions, (iv) Pylorus, gall-bladder, and stomach.
   b. Diffuse.
4. CHRONIC PROLIFERATIVE PERITONITIS.
   a. Local, e.g., sugar-ice liver (Zuckergussleber).
   b. Diffuse.
   c. Polyserositis, polyorrhomenitis, or Concato's disease.

General Etiology—Types (3) and (4) form an extremely difficult group of cases, further complicated by multiplicity of names. *Spread of bacterial inflammation* is an undoubted factor in certain cases, e.g., pelvic peritonitis, diverticulitis. Pathological changes of similar type, but of varying extent, may also occur in perisplenitis, perihepatitis, etc., and in diffuse peritonitis. At the other extreme is polyserositis, inexplicable as spread of ordinary inflammation, but with pathological changes similar to those which occur locally, e.g., 'sugar-ice liver'. Further, changes originally local tend to spread gradually over peritoneum.

The problem is complicated by frequent impossibility of deciding in given case, e.g., a sigmoid tumour, (1) to which type it belongs, (2) whether it is tuberculous. All forms tend to spread from site of origin.

Pericolitis, pericolitis sinistra, perisigmoiditis, hyperplastic pericolitis, are synonyms for various local types.

CHRONIC ADHESIVE PERITONITIS.

Causes.—Include : (1) Ulceration of gut, not necessarily perforating ; (2) Spread through lymphatics of inflamed organs, or through diaphragm in pleurisy ; (3) Irritation of foreign bodies.

Varieties—

LOCAL TYPE.—Common forms : (1) Pelvic peritonitis ; (2) Around liver and spleen ; (3) Diverticulitis and pericolitis ; (4) Around pylorus, gall-bladder, and stomach.

1. PELVIC PERITONITIS.—From inflammatory diseases of pelvic organs.

   Chronic Hæmorrhagic Peritonitis.—Rare. Vascular new fibrous tissue present : hæmorrhages occur and organize : perhaps comparable to hæmorrhagic pachymeningitis. Usually localized to pelvic peritoneum.

2. LIVER AND SPLEEN.—Adhesions common, mainly to diaphragm : found at autopsy. No symptoms known.
3. DIVERTICULITIS.—*See below*
4. PYLORUS, GALL-BLADDER, AND STOMACH.—Extent variable. With gall-stones may be marked, pyloric thickening and adhesions. Sometimes peptic ulcer present, not necessarily perforated ; or catarrh of stomach. *Adhesions round stomach* may cause dyspepsia and vague pains. Division at operation not always curative, owing to recurrence. Adhesions to liver common.

ADHESIONS OF SMALL INTESTINES.—Usual site, lower ileum (Lane's ileal kink).

DIFFUSE TYPE.—Widespread adhesions may be present : apparently with origin similar to localized forms.

Note.—In the more chronic cases, differentiation of either local or diffuse forms from chronic proliferative, tuberculous, or carcinomatous peritonitis is often impossible. (*See* CHRONIC PROLIFERATIVE PERITONITIS for symptoms and signs.)

Diverticulitis.*—As a clinical term, this applies to pathological processes resulting from acquired false diverticula of the large intestine.

ETIOLOGY.—*Age* : middle age or later. *Males* commoner than females. Usually stout individuals with chronic constipation.

MORBID ANATOMY.—Size of cavity small, up to a French bean ; aperture often minute. Usually multiple, may be numerous. *Site* : Great majority in lower portion of large intestine, especially sigmoid flexure ; sometimes scattered throughout

* Maxwell Telling, *British Journal of Surgery*, 1917, Jan.

Diverticulitis—Morbid Anatomy, *continued.*

colon; occasionally localized in other sites, e.g., cæcum. Origin usually opposite the appendices epiploicæ, which they tend to enter. As lumen enlarges, apex curves backwards towards the attachment of the mesentery.

PATHOGENESIS.—Chronic constipation is most important factor. Influence of flatulent distention is doubtful.

SYMPTOMATOLOGY.—Very varied. Diverticulum tends to enlarge, to contain fæcal matter, and to undergo and to produce various secondary processes on which the symptoms depend. These secondary processes result from various grades of inflammation, and may be combined in various manners. The principal results are:—

1. ACUTE DIVERTICULITIS AND INFLAMMATORY DISTURBANCES.—Due to ulceration and perforation. Commonest type. *Symptoms*: Pain, tenderness, and rigidity in left lower quadrant, with or without a tumour. Bladder symptoms not uncommon. Closely resembles appendicitis, but on left side, and similarly may be acute, subacute, recurrent, or chronic. *Localized abscess formation* not uncommon, with fever and leucocytosis. Symptoms may be referred to pelvic organs, especially in females, suggesting tubo-ovarian disease. General peritonitis uncommon, owing to adhesions and tracking of diverticulum towards mesenteric attachment. Onset of acute symptoms may be sudden, and follow trauma, e.g., straining at stool, enema, sudden exertion. Other sequelæ below may develop subsequently.

2. ADHESIONS to various structures.—Results may be: (i) Various vague pains and constipation; vesical and pelvic syndromes. (ii) *Fistulæ*, due to adhesions to organs and perforation; diverticulitis is commonest cause of vesicocolic fistulæ (commoner than cancer), and operation is often successful. (iii) Acute intestinal obstruction by bands or kinks (iv) Local abscesses.

3. PERIDIVERTICULAR FIBROUS HYPERPLASIA (chronic diverticulitis).—From leakage of toxins or bacteria through walls. The fibrous tissue may be an inch or more in thickness; firm tumour forms; general results resemble cancer. Contraction of fibrous tissue stenoses the gut, producing chronic obstruction. Chronic proliferative peritonitis may develop, result resembling 'tuberculous cæcal tumour.'

Cancer may develop in the tumour.

Tumours are present in 30 per cent of all forms, and tend to vary in size from time to time.

DIAGNOSIS.—Diverticulitis is a possibility in all patients over middle age with inflammatory troubles in left lower quadrant, in all cases suggesting carcinoma of colon, and in vesico-

intestinal fistulæ; it must be excluded before deciding that the condition is inoperable. Bismuth meals and X rays are often decisive.

Diagnosis from Carcinoma.—(1) Absence of wasting and cachexia—patients usually stout; (2) Long history of abdominal pain in left lower quadrant; (3) Persistent absence of blood from stools; (4) Bismuth meals and X rays; (5) Sigmoidoscopy negative; (6) Pyrexia and leucocytosis may be present.

TREATMENT.—Operative. Other diverticula must be looked for, and obliterated if found. Gut must be handled carefully. If symptoms are mild or operation is contra-indicated, may be treated with repeated enemata, liquid paraffin, etc., as in chronic constipation; but treatment must be very careful, and operation is the correct procedure.

Jackson's Membrane.—A fine membrane surrounding the cæcum but of varying extent: usually almost transparent, but occasionally opaque. Congenital in origin, probably an extension of the omentum carried down in descent of the cæcum (Gray and Anderson).

CHRONIC PROLIFERATIVE PERITONITIS.

SYNONYMS.—Chronic indurative, hyperplastic, or adhesive peritonitis. *For generalized cases* affecting all serous membranes, mediastinum, etc.: Polyorrhomenitis, polyserositis, Concato's disease. *For local forms*: Proliferative perisplenitis or perihepatitis, sugar-ice liver, Zuckergussleber, etc., depending on organ affected.

Polyorrhomenitis, polyserositis, or Concato's disease is a widespread affection of peritoneum, pleura, pericardium, and mediastinum. Symptoms and physical signs of chronic proliferative peritonitis, pericarditis, etc., are combined. In early stages, generally very obscure.

Pathogenesis.—In generalized *polyorrhomenitis*, causal factors unknown: usually young subjects. Theories are: (1) Idiopathic overgrowth of fibrous tissue. (2) Tuberculous: often suggested, but no proof and no typical changes present, histological or bacteriological. (3) Spread from a local origin: certainly, local proliferative forms essentially tend to spread; also after repeated tapping for ascites, proliferative peritonitis may occur round site of punctures. There are also various inconclusive theories of 'toxic' action, e.g., from interstitial nephritis, products of pyogenic organisms, lead (unsupported).

Of local forms, *perihepatitis* (sugar-ice liver) is most frequent; usually about middle-age, commonly (not always) associated with chronic alcoholism. Is often accompanied by varying grades of more diffuse peritonitis and interstitial nephritis.

Thus, alcohol is a certain factor in some cases, but insufficient to account for all; tuberculosis is unproved, and other factors are unknown.

A succession of able Guy's physicians, from Addison to Hale White, have held that recurrent ascites in chronic alcoholism

Chronic Proliferative Peritonitis—Pathogenesis, *continued.*

is always due to peritonitis, and that with ascites purely due to alcoholic cirrhotic liver, life is not prolonged sufficiently for recurrence. Also they hold that perihepatitis of 'sugar-ice' type, with absence of jaundice, is definitely associated with, and the sequel of, interstitial nephritis.

Morbid Anatomy.—Extent and distribution very variable. Of changes described below, almost every combination occurs of the local and general type.

PERITONEUM.—Greatly thickened from fibrous tissue, ¼ to ½ inch; glistening white; distribution irregular: areas of cartilaginous hardness; much contracted. *Omentum*: rolled transversely across abdomen, from thickening and contraction, especially on left side. *Mesenteries shortened*: intestines drawn against spine, with lumen narrowed and length shortened. In absence of fluid, may form a palpable irregular mass. Occasionally *pigmented streaks and patches*. Non-tuberculous nodules.

ADHESIONS.—Variable, local or general. Of all degrees, but often slight. Kinking of intestines may result. May be areas with organs involved in dense adhesions, e.g., pylorus to liver, gall-bladder and pancreas, cæcum and appendix, sigmoid.

EFFUSION.—Variable, *from slight to enormous*. Depends only partly on amount of adhesions. Occasionally 'chylous'.

LOCAL CHANGES OVER ORGANS.—Liver, spleen, etc., may be merely adherent, e.g., to diaphragm, as part of general peritonitis. In other cases thickening is mainly confined to certain sites, but changes are similar in character to general type.

LIVER.—'Sugar-ice liver' (Zuckergussleber): *perihepatitis*. Organ contracted, but thick capsule may strip easily. Liver substance often remarkably normal: some fibrosis (possibly spread from covering, or due to pressure), but advanced cirrhosis rare. Gastrohepatic omentum and portal vein may be constricted (whence ascites). Usually spleen and to some degree general peritoneum affected: also *interstitial nephritis*. Symptoms: (*a*) Similar to ordinary 'alcoholic cirrhotic liver', if change local: only distinguishable at autopsy. (*b*) Part of diffuse type or polyorrhomenitis.

SPLEEN.—Perisplenitis. Similar 'iced' spleen occurs, but rare unless liver also affected.

PYLORUS, GALL-BLADDER, LIVER, STOMACH, AND PANCREAS.—Dense adhesions may involve these, especially pylorus ('perigastritis').

CÆCUM AND APPENDIX.—May be indistinguishable from the 'tuberculous cæcal tumour' (*see* p. 164).

SIGMOID.—Resembles chronic fibrous diverticulitis.

Symptoms and Signs in Diffuse Type.—Obscure. Variable with: (1) Extent; (2) Site of lesions; (3) Relation of effusion and adhesions. Intervals of comparative freedom. The most constant are:—

ABDOMINAL PAIN.—Variable and intermittent. No case is entirely free.

GASTRO-INTESTINAL DISTURBANCE.—Troublesome constipation, occasionally diarrhœa and vomiting, due to stenosis, kinking, and adhesions. *Anorexia, flatulence,* and *dyspepsia* common.

WEAKNESS AND PROGRESSIVE WASTING.

Variable: Pyrexia and rapid pulse; dyspnœa and respiratory symptoms (depend on thoracic changes). Occasionally: Œdema or thrombosis of legs; difficulty in micturition. Jaundice rare.

ABDOMINAL SIGNS.—

INSPECTION.—*Irregular and variable distention* (fluid and meteorism). Skin dry. Veins distended.

PALPATION.—Increased and doughy resistance. Various masses and tumours.

PERCUSSION.—Fluid, which may be encysted and not movable. Irregular resonant areas.

Friction sound rare.

PROGRESS.—Insidious advance. Duration, usually years.

Symptoms and Signs in Local Type.—Depend on site: resemble local chronic tumours from other causes.

Diagnosis.—By long observation only. From chronic peritonitis of tuberculosis, of extension of inflammation, and from carcinoma, often impossible even at operation.

Treatment.—Symptomatic. *Paracentesis* when indicated: often repeated on enormous number of occasions. For definitely localized intestinal tumours, excision or various short-circuiting operations for temporary relief. Progressive nature generally contra-indicates operation. The dense adhesions and thickened peritoneum render operations prolonged, difficult, and usually unsatisfactory.

IV. NEW GROWTHS IN THE PERITONEUM.

Varieties.—(1) Benign neoplasms: fibroma, lipoma, myoma, angioma; all very rare. (2) Primary malignant neoplasms (sarcoma). (3) Secondary malignant neoplasms (carcinoma). (4) Cysts. Also: tuberculosis.

PRIMARY MALIGNANT NEOPLASMS.

HISTOLOGY.—*Sarcoma.* Growths formerly considered 'carcinoma' now interpreted as endothelioma or mixed carcinomatous sarcoma.

SARCOMATOSIS OF PERITONEUM: DISSEMINATED MILIARY NODULES.—Very rare. The retroperitoneal glands may enlarge, but viscera escape.

Very rarely: sarcoma in omentum and mesentery.

RETROPERITONEAL SARCOMA.—Not strictly a peritoneal neoplasm: origin from retroperitoneal connective tissue. At any age, especially under 5 years (next to tuberculosis, the commonest neoplasm of infancy). Immobile tumour, extends

Primary Malignant Neoplasms, *continued.*

forward near mid-abdomen, usually crossed by coil of gut; hence variations in resonance: hard, but pseudo-cysts common. No ascites. Constitutional symptoms of neoplasm. Local symptoms vary with size and extent.

SECONDARY MALIGNANT NEOPLASMS.

HISTOLOGY.—Almost invariably *carcinoma.* (Peritoneum escapes in great proportion of abdominal neoplasms.)

PRIMARY GROWTH.—(1) In *ovaries* most frequent; (2) *Pylorus,* stomach, intestines, gall-bladder. Very rarely in breast, or œsophagus.

SEX.—Commoner in women. After middle age.

VARIETIES.—

1. DISSEMINATED MILIARY NODULES.—Carcinomatosis of peritoneum. Size: from pin's head to pea. Often great effusion, masking physical signs. Peritoneum, in slow cases, may show changes described as proliferative peritonitis.
   Characteristics in very chronic forms are: (i) Great thickening and contraction of the peritoneum; (ii) Rolled transverse omentum; (iii) Contracted mesentery and fixed intestines; (iv) Various adhesions and effusions.
2. MASSES OF GROWTH.—Miliary nodules and changes as in previous type may also be present.
3. COLLOID CANCER.—Secondary to tumour in ovary or stomach. Sometimes possibly a primary growth (from Wolffian body). Attains enormous size. Masses palpable. No effusion.

EFFUSION.—Serous, hæmorrhagic, or 'chylous'. *Cytology*: endothelial cells; may be marked mitosis; diagnostic value doubtful.

DURATION.—Rarely exceeds six months from recognition.

DIAGNOSIS.—General characteristics: wasting, with recurrent ascites; after tapping, masses may be palpable. Diagnosis aided by: (1) Local primary tumours; (2) After middle age, large masses are usually cancer; (3) Inguinal glands or umbilical nodules. In hepatic cirrhosis, jaundice and enlarged veins are present, but diagnosis may be impossible, as also from tuberculosis and forms of chronic peritonitis.

CYSTS OF PERITONEUM.

Abdominal neoplasms frequently become cystic. Numerous other cysts occur: (1) Mesenteric cysts. (2) Dermoids and teratomata: mesenteric or retroperitoneal. (3) Urachal cysts. (4) Parasitic cysts: hydatids; very rarely cysticercus (no symptoms).

Mesenteric Cysts.—

ORIGIN.—Doubtful: possibly embryonic from remains of Wolffian body, or of intestinal epithelium.

MORBID ANATOMY.—In mesentery of small intestine: may be sessile and attached to intestine. Usually unilocular. Epithelium or fibrous-tissue lining. Contents: (1) *Serous*: contain albumin, cholesterin, and sometimes mucin. (2) *Chylous*: not uncommonly contain true chyle. (3) Hæmorrhagic: rare. Also (4) Hydatid, and (5) Dermoid.

PHYSICAL SIGNS.—(1) *In middle line*, near umbilicus. Usually more on right. (2) Round, definite outline, smooth and regular (except hydatid), tense: may fluctuate. (3) *Great mobility*, in circular directions, but especially side-to-side. (4) *Dull, with resonant area in front*, from coil of intestine. Size, few inches upward. Large cysts often fixed by adhesions, and completely dull.

DURATION.—Many years.

SYMPTOMS.—Often slight. Pain and constipation from enlarging size; occasionally gastro-enteritis; rarely acute obstruction. *May suppurate.*

DIAGNOSIS.—Very difficult, especially from ovarian and from renal tumours. Large fixed cysts resemble pancreatic cysts, retroperitoneal cysts, and other fixed tumours.

Omental Cysts.—Very superficial and extremely mobile.

Retroperitoneal Cysts.—In retroperitoneal tissues. Position resembles mesenteric cysts, but fixed. No diagnosis possible from pancreatic cysts and fixed mesenteric cysts.

Urachal or Allantoic Cysts.—Rare. Origin from incomplete obliteration of urachus between bladder and umbilicus. In men resemble full bladder, but not removed by catheter. In women (rarer) resemble ovarian cyst. *Often become malignant.*

Treatment of Cysts.—Laparotomy and removal.

V. ASCITES.

(*Hydro-peritoneum.*)

The accumulation of *non purulent* fluid in the peritoneal cavity.

Etiology.—Due to local obstruction of the portal system, or to certain general conditions affecting the circulation in which pleural and other effusions may occur.

LOCAL CAUSES.—

PORTAL OBSTRUCTION (Portal vein or main tributaries).—(1) Terminal branches in liver: portal cirrhosis of liver; chronic passive congestion; syphilis. (2) Compression in the gastrohepatic omentum and hilus: enlarged glands (malignant, tuberculous, Hodgkin, etc.); neoplasms. Rare: perihepatitis and local chronic peritonitis; aneurysms.

CHRONIC PERITONITIS (*see* p. 496).—Tuberculosis, neoplasm, adhesive and proliferative forms. Hydatid cysts.

THROMBOSIS OF PORTAL VEIN.

SPLENIC ANÆMIA AND BANTI'S DISEASE.—Probably from disease of veins of portal system.

TUMOURS.—Especially solid ovarian tumour.

Ascites—Etiology, *continued.*

GENERAL CAUSES.—

CARDIAC FAILURE.—Cardiac, pulmonary, or arteriosclerotic.

NEPHRITIS.—Especially chronic parenchymatous form.

Common causes: Portal cirrhosis of liver. Cardiac failure. *Not infrequent*: Chronic parenchymatous nephritis. Tuberculous peritonitis (especially in children). Carcinoma of liver.

Note. Malignant disease of liver, pancreas, etc., often produces ascites by action of enlarged glands in hilus of liver. Syphilis probably acts by presence of peritonitis. For the methods in which the various causes produce ascites, see also under the diseases separately.

Symptoms.—Progressive uniform abdominal enlargement. Various results of pressure on diaphragm and interference with thoracic and abdominal organs, depending on rapidity of formation rather than quantity. Fluid may be absorbed and return.

Physical Signs.—

INSPECTION.—Varying distention, commencing in flanks. When effusion large: skin tense; lineæ albicantes; navel prominent; superficial veins distended, flow from below up (extreme in portal thrombosis). Veins round navel distended; caput medusæ, especially in hepatic cirrhosis.

PALPATION.—'Fluid thrill' transmitted through abdomen. A solid organ or tumour is palpated through a layer of fluid by 'dipping' with tips of fingers.

PERCUSSION.—(1) 'Shifting dullness'. Percuss on back, and then on side. For small quantities, try knee-elbow position and percuss near navel. (2) Flanks dull, centre resonant. In large effusions, general dullness.

Diagnosis.—(1) Shifting dullness (pathognomonic of effusion when obtained). (2) Fluid thrill. (3) Signs of portal-peripheral anastomoses (for routes, *see* CIRRHOSES OF LIVER, p. 474). Diagnosis from:—

OVARIAN TUMOUR.—Dullness central, resonance lateral. Examination per vaginam may decide when doubtful.

LARGE HYDATID ('hydatid thrill'); PANCREATIC CYSTS.—Diagnosis may be impossible.

Peritoneal Fluid in Ascites.—(*See also* PLEURAL FLUIDS.)

SEROUS.—Clear light yellow; is usual type. Specific gravity: transudates, e.g., nephritis, under 1015; exudates (peritonitis) over 1015. (As regards diagnosis, the commonest specific gravity, unfortunately, appears to be 1015.) Albuminous. Occasionally clots spontaneously.

HÆMORRHAGIC.—In tuberculosis (commonest cause): cancer (highest relative percentage). Rare in cirrhosis. Also occurs in ruptured tubal pregnancy.

OPALESCENT.—

1. TRUE CHYLOUS.—Yellowish opacity due to fat, which forms layer on surface, cleared by ether. Rare. Occurs in affections of thoracic duct and lymphatics, also filariasis.

2. PSEUDO-CHYLOUS.—Opalescence due to lipoid, soluble in alcohol, not in ether; small amount of true fat present; varies at different tappings. Occurs in all forms, especially chronic parenchymatous nephritis. Prognosis bad.

CYTOLOGY.—Cells often difficult to distinguish, owing to degeneration.

1. SMALL LYMPHOCYTES.—In tuberculosis.
2. 'ENDOTHELIAL' CELLS.—Large cells with nucleoli. Predominant in passive and neoplasmic effusions, but usually many present in all effusions.
3. CARCINOMA CELLS.—Extremely rare. Various nuclear changes, e.g., marked mitosis, are described, but rarely are reliable.

Treatment.—

MEDICAL.—Depends on cause. Aperients should be used only in moderation. Restriction of fluids, diuretics, salt-free diet, of little general value.

PARACENTESIS.—

INDICATIONS.—(1) Abdominal: great distention, pain, and alimentary disturbance. (2) Thoracic, from displacement of diaphragm: dyspnœa, collapse at bases, and cardiac disturbance.

TECHNIQUE. (1) Empty bladder. (2) Choose site of puncture, mid-line above pubes (must be dull on percussion). (3) Inject 1 to 2 per cent novocain ad lib. (4) Sterilize skin with iodine. (5) Small incision through skin. (6) Trocar and cannula, medium size, plunged into peritoneum, trocar withdrawn, and fluid allowed to flow slowly, cannula strapped to skin with plaster. (7) Binder round abdomen, tightened at intervals. Fluid flows for several hours.

Trocar and cannula: Fasten rubber tubing to cannula, then pass trocar through wall of tubing and down cannula; after insertion in peritoneum and withdrawal of trocar, fluid flows through tubing into receptacle placed on floor.

Alternative site (used after repeated punctures): Midway between the anterior superior spine and umbilicus; may perforate deep epigastric artery, causing serious hæmorrhage entailing ligature.

TALMA-MORISON OPERATION ('EPIPLOPEXY').—Artificial anastomosis of portal and general circulation: (a) Rub surfaces of liver and diaphragm to obtain adhesions; (b) Suture portion of omentum in sheath of rectus. Only justifiable with short history and rapid ascites. Results doubtful. (Anastomosis often established by nature.)

Section VI.—DISEASES OF THE RESPIRATORY SYSTEM.

CHAPTER LXXX.

DISEASES OF THE NOSE.

I. HAY FEVER.

An affection of the upper air passages and conjunctiva, due to hypersensitiveness to proteins of pollen of certain plants

Protein hypersensitiveness is discussed under bronchial asthma, with which hay fever is closely allied and attacks often interchangeable In Europe, hypersensitiveness in hay fever is solely to pollen of grasses, in America also, in fall, to pollen from ragweed. tested by conjunctival reactions to extracts of pollen

AGE —Commonest in young adults Diminishes with age

SEX More frequent in women

Symptoms.—

RESEMBLE SEVERE CORYZA -

1. Sneezing fits
2. Conjunctival irritation and lachrymation
3. Nasal discharge, watery, copious and continuous
4. Headache and general depression

Cough not uncommon

GENERAL CONGESTION OF NASAL MUCOUS MEMBRANE present Nasal discharge may be most prominent symptom (paroxysmal rhinorrhœa).

Duration.—Days to weeks. Depends on exposure to pollen

Treatment.—

1. ACTIVE IMMUNIZATION —Increasing injections of extract of pollen. Extract of pollen of one grass protects against all timothy grass (*Phleum pratense*) commonly used Results very good. Immunize before attack commences
2. PASSIVE IMMUNIZATION —Dunbar's pollantin, specific antiserum · locally applied to nose and conjunctiva before rising Partially effective.

Sea voyage or prolonged absence from causal pollen may produce desensitization : tends to return.

NASAL TREATMENT —Remove polypi and correct slight abnormalities, but extensive operations inadvisable Cauterization of septum often of considerable effect.

II. EPISTAXIS.

(Bleeding from the Nose.)

Etiology.—Causes are: (1) Local; (2) General.

1. LOCAL CAUSES.—Trauma; picking nose; insertion of foreign bodies; neoplasms, nasal, antral, etc. Rare: hereditary multiple telangiectases.
2. GENERAL CAUSES.
   - AT PUBERTY.—Especially in delicate children.
   - ACUTE SPECIFIC FEVERS.—Onset of enteric, scarlet fever, and occasionally measles.
   - CONDITIONS WITH HIGH BLOOD-PRESSURE. Arteriosclerosis, nephritis, gout, cirrhotic liver. Often precedes or indicates liability to apoplexy.
   - BLOOD DISEASES.—All severe anæmias, hæmophilia.
   - ALTERATIONS OF ATMOSPHERIC PRESSURE.—E.g., occurs in mountaineering.
   - IN SUPPRESSION OF MENSES.—Rarely.

PROBABLE CAUSE ACCORDING TO AGE:—

- CHILDHOOD.—Trauma, picking nose, foreign bodies, acute specific fevers.
- PUBERTY.—Spontaneously.
- MIDDLE AGE.—Blood diseases, neoplasms.
- AFTER MIDDLE AGE.—High blood-pressure, neoplasms.

Diagnosis.—Occasional difficulty if blood be swallowed and vomited, or, rarely, coughed up.

Prognosis.—Rarely serious: tends to clot. Death extremely rare.

Treatment, if necessary:—

- HÆMOSTATICS: Adrenalin (1–1000), perchloride of iron applied to mucous membrane.
- COLD WATER or ICE to bridge of nose.
- PLUG NARES, if serious. Hot-water bottles to feet: or legs to knees in hot water.

CHAPTER LXXXI.

DISEASES OF THE LARYNX.

I. ACUTE CATARRHAL LARYNGITIS.

Etiology.—

EXCITING CAUSES:—

1. COLD.
2. OVER-USE OF VOICE.
3. ACUTE SPECIFIC FEVERS.—Common in measles, influenza, small-pox.
4. LOCAL IRRITANTS.—Gases, hot liquids, foreign bodies.

Acute Catarrhal Laryngitis—Etiology, *continued.*

PREDISPOSING CAUSES.—Gout, alcohol and tobacco in excess, and possibly rheumatism.

AGE.—None immune, through cause varies. More serious in children owing to narrow glottis.

Morbid Anatomy.—Laryngoscope shows: mucous membrane of ary-epiglottidean folds congested, cords red and swollen, mobility often impaired, some mucus.

Symptoms—

ORDINARY ATTACK IN ADULTS. (1) Tickling in larynx, irritated by cold air. (2) Voice husky. (3) Dry cough; slight mucoid sputum, often streaks of blood. (4) Constitutional symptoms mild: slight pyrexia.

SEVERE ATTACK. Voice entirely lost; swallowing painful; pain over larynx. Dyspnœa rare.

Diagnosis.—Rarely difficult. Nervous aphonia may be distinguished by laryngoscope.

Prognosis.—Never fatal. Prognosis for voice often important: may be permanently impaired. If not treated, chronic laryngitis may follow.

Treatment.—

GENERAL.—

WARM, MOIST ROOM, with much FRESH AIR: temperature 60 to 65°.

DIET.—Light. Warm drinks. If dysphagia, *semi-solids* (custards, etc.), usually less painful than fluids. Sucking ice often eases.

ACETYLSALICYLIC ACID gr. x, t.d.s., or diaphoretic mixture.

BOWELS OPENED FREELY: calomel gr. ij and salines

TINCT. ACONITI ♏j in water, hourly, for 6 doses, if high temperature (but watch pulse: stop if this weakens).

TONIC during convalescence.

LOCAL.—

a. EXTERNAL—Antiphlogistine, mustard leaf, cold compress or ice-bag (cold generally relieves best).

b. INHALATION.—Tinct. benzoini co. ʒj in pint of water at 140° F.

c. SPRAY.—In oil atomizer, 5 per cent solution of menthol in paroleine (Semon).

d. LOZENGES.—Troch. menth. c. krameria, or cocain c. krameria.

II. CHRONIC LARYNGITIS.

Etiology.—Often chronic from onset; or follows repeated acute attacks. Over-use of voice is common factor. Alcohol and tobacco in excess often accessory.

Symptoms.—

1. ALTERATION OF VOICE and hoarseness: voice tires rapidly.
2. TICKLING IN LARYNX, with desire to cough.

Laryngoscope: Mucous membrane swollen; vocal cords thickened, mucus on surface. Hyperæmia slight. May be weakness of adductor muscles.

Diagnosis.—Laryngoscopic examination in prolonged cases. Tuberculous, malignant, and syphilitic laryngitis may commence as chronic catarrh.

Prognosis for Voice.—Often permanently impaired. May be resistant to treatment.

Treatment.—Examine nostrils for obstruction. *Rest voice.* Avoid hot rooms, loud speaking, alcohol and tobacco. Same treatment as for acute attacks may be given. Local application to larynx, zinc chloride (gr. xx to ℥j), with laryngeal brush, alternate days for three weeks. Massage to larynx. At Mont Dore, spa treatment is organized, with massage, vibration, and ionization.

III. ŒDEMATOUS LARYNGITIS.

(*Œdema of the Glottis.*)

Very serious, owing to rapid asphyxiation and death. Never primary; secondary to local or general conditions.

Etiology. Causes are:—

1. LOCAL.—
   - *a.* TRAUMA.—Sharp foreign bodies, scalds, etc
   - *b.* SEQUEL TO ACUTE LARYNGITIS.
   - *c.* SEQUEL TO CHRONIC LARYNGITIS.—Tubercle or syphilis.
   - *d.* LOCAL INFLAMMATORY CONDITIONS (rarely).—Cellulitis of neck, erysipelas, diphtheria.
2. GENERAL.—
   - *a.* NEPHRITIS, chronic or acute.
   - *b.* ANGIONEUROTIC ŒDEMA.
   - *c.* ACUTE INFECTIOUS FEVERS (rarely).

Symptoms.—

DYSPNŒA.—Sudden onset, rapidly increasing. May be inspiratory stridor. Voice disappears.

ON EXAMINATION.—Epiglottis greatly swollen, can be seen and felt; ary-epiglottidean folds swollen and may meet. Œdema may be subglottic. True vocal cords rarely affected.

Diagnosis.—By sudden dyspnœa and swollen epiglottis.

Treatment.—Ice to suck and to neck. Air moist. If severe, spray with cocaine 20 per cent. and scarify epiglottis. *Tracheotomy* without hesitation; in absence, mortality high.

IV. TUBERCULOUS LARYNGITIS.

Etiology.—Very rarely primary. Practically always secondary to pulmonary tuberculosis, though disease of larynx often advanced with but slight signs at apex

Tuberculous Laryngitis, *continued.*

Morbid Anatomy.—Commences at posterior extremities of ary-epiglottidean folds, and on interarytenoid folds, and tends to spread forwards.

ON EXAMINATION

FIRST STAGE: Mucous membrane pale, thickened, and infiltrated.

SECOND STAGE: Tuberculous masses (rarely seen).

THIRD STAGE: Ulceration—broad, shallow, gray, covered with exudation. General appearance 'worm-eaten.'

DISEASE SPREADS: (1) Forwards to epiglottis, which may be destroyed, (2) By ulceration, causing perichondritis and necrosis of cartilages. Vocal cords thickened. Less often it spreads backwards to pharynx. Rarely, stenosis of larynx results.

Symptoms.—

ONSET. Slight huskiness of voice and irritation. Later, hoarseness and aphonia.

COUGH.—As ulceration increases.

DYSPHAGIA.—Especially with ulceration of epiglottis or spread to pharynx. May be agonizing.

Diagnosis.—Based on: (1) Laryngoscope pallor, infiltration, and ulceration; (2) Pulmonary tuberculosis; (3) Bacilli in sputum.

Diagnosis from:—

1. SYPHILITIC LARYNGITIS.—Usually painless. Laryngoscope: more congestion, commences *at base of epiglottis*, ulceration deep. Scarring common.
   Syphilis and tubercle may co-exist.
2. CARCINOMA.—Papillary growth from vocal cords or ventricular bands; unilateral in early stage.

Treatment.—*Complete silence for many months.* Spray throat with menthol and olive oil, or 'spirone' (Churchill's inhalant—KI in acetone, glycerin, and water). Recovery in early stages.

If general condition good, ulcers should be cauterized or removed. An ulcerated epiglottis may be removed (relieves dysphagia), but laryngeal condition may progress more rapidly afterwards.

PAIN.—Insufflation of orthoform, gr. v to x, half an hour before meals.

DYSPHAGIA.—Food semi-solid. Spray with cocaine, or orthoform insufflation, before food. Wolfenden position: head hangs over bed, and food is sucked through tube.

V. SYPHILITIC LARYNGITIS.

Of frequent occurrence.

Etiology.—

CONGENITAL SYPHILIS.—(1) In first six months or early years as catarrhal laryngitis; (2) At puberty as in tertiary syphilis.

SECONDARY SYPHILIS.—Resembles acute laryngitis, but very resistant. Occasionally ulcerates. Condylomata very rare.

TERTIARY SYPHILIS.—(1) True gumma; commences at base of epiglottis; results in (*a*) stenosis of larynx—may be extreme, (*b*) deep ulceration—less common. (2) Diffuse infiltration.

Symptoms.—Chronic laryngitis. Hoarseness. Cough rare. Almost invariably painless.

Treatment.—Antisyphilitic. Rapid relief with potassium iodide, though scarring may follow.

STENOSIS.—Dilatation by Schrötter's bougies may relieve, but recurrence usual. Tracheotomy may be necessary.

VI. LARYNGISMUS STRIDULUS.

Idiopathic spasm of the glottis, no inflammation present. Confined to children.

Etiology.—

AGE.—About 18 months. Not under 6 months, rarely over 3 years

RICKETS very common. Frequently associated with carpopedal spasms and tetany.

EXCITING CAUSE.—Scolding, or any irritation.

Symptoms.—Onset at night or early morning. No cough or hoarseness present. Respiration ceases, period of apnœa: the child struggles for breath; becomes congested; seizure terminates with crowing inspiration as spasm relaxes. Attacks at first occasional, may become very frequent.

Prognosis.—Rarely, but occasionally, fatal.

Treatment.—

DURING SPASM.—Throw cold water on the face, or tickle fauces to relieve spasm; or hot sponge on larynx. If necessary, a little chloroform.

FOR RECURRENT SPASMS.—Place in hot bath, and sponge back and chest with cold water.

FURTHER TREATMENT.—(1) Chloral (syr. chloral ℿx, t.d.s.); (2) Treat rickets; (3) Regulate bowels; (4) Keep child quiet and avoid excitement and irritation.

Diagnosis in Conditions of Spasm or Obstruction of the Larynx.

1. LARYNGISMUS STRIDULUS.—Not under 6 months; no previous cough or hoarseness; onset sudden; definite period of cessation of respiration ('holding the breath'). Rarely fatal.
2. CONGENITAL LARYNGEAL STRIDOR.—Congenital; continuous; ceases after few months; no distress. Never fatal.
3. CATARRHAL SPASM OF THE LARYNX.—Previous slight cough or hoarseness; onset rapid but not sudden; no cessation of respiration; attacks intermittent. Never fatal.
4. CATARRHAL LARYNGITIS (OR ACUTE LARYNGITIS).—Previous cold, dyspnœa, and fever; dyspnœa progressively increases. Longer duration, no intermissions. Dangerous. May be simple or diphtheritic. (*See* DIPHTHERIA, p. 43).

Laryngeal Spasm or Obstruction—Diagnosis, *continued.*

5. WHOOPING-COUGH.—Previous cough. Paroxysm commences with short expirations before inspiration and whoop.
6. IN PRESENCE OF ADENOIDS OR ENLARGED TONSILS, cough may suggest inspiratory stridor and laryngeal obstruction.
7. PAPILLOMA OF LARYNX.—Diagnosis by laryngoscope only. Symptoms of chronic laryngitis.

VII. CATARRHAL SPASM OF THE LARYNX.

(*Spasmodic Laryngitis. Spasmodic Croup.*)

Spasm of the larynx occurring with mild laryngitis. (Acute of catarrhal laryngitis applies to condition sufficient to cause obstruction in absence of muscular spasm.)

Etiology.—

AGE.—Two to four years. Rare under six months.
ADENOIDS and ENLARGED TONSILS.—May be present.
EXCITING CAUSE.—Chill. Indigestion.

Symptoms.—Previous slight cough. Dyspnœa and barking character of cough increase at night until child awakes with
ATTACK.—Respiration oppressed, crowing inspiration and croupy cough, husky voice, struggles for breath; signs of laryngeal obstruction, viz., recession of epigastrium and suprasternal fossa during inspiration; appears serious, child and parents terrified. Cessation rapid and child sleeps. Duration half to three hours.
Attack recurs for two or three nights. Child fairly well during day. Never fatal.

Treatment.—

IMMEDIATE.—Emetic of vin. ipecac. ʒj or pulv. ipecac. gr. x, every quarter-hour till vomiting. Steam kettle. Heat to larynx or hot bath. A little chloroform if necessary.
TO PREVENT RECURRENCE.—Vin. ipecac. ♏iij t.d.s. during day. Phenazone gr. ij at night. Avoid chills, but have fresh air in room.
LATER.—Treat adenoids if present.

CHAPTER LXXXII.

DISEASES OF THE BRONCHI.

I. ACUTE BRONCHITIS.

Acute catarrhal inflammation of mucous membrane of trachea and bronchi, large and small. In smaller bronchi, constitutes capillary bronchitis (*see* BRONCHOPNEUMONIA, p. 66).

Etiology.—

AGE.—None immune. Frequent and serious in old people and children (dentition, rickets, and specific fevers).

SEASON.—Common at change of seasons. May recur yearly.

CHILL.—Often from downward spread of coryza (i.e., 'cold on the chest').

SOME PREDISPOSING FACTORS IN CASES OF 'CHILL.'—(1) Hereditary predisposition: some persons and families 'catch cold easily.' (2) Infectivity and epidemicity: occurs even apart from *B. influenzæ*. (3) Occupations: dust, hot atmospheres, sedentary occupations.

ONSET OF SPECIFIC FEVERS.—Constant in measles and whooping-cough. Rarely absent in enteric.

DEBILITATING CONDITIONS.—Nephritis, heart lesions, diabetes, gout, rickets, etc. Subacute attacks frequent.

IRRITANT GASES.—Chlorine, nitric acid fumes, etc.

Bacteriology.—Common: pneumococcus; also streptococcus, *M. catarrhalis*, *B. influenzæ*. Uncommon: *B. coli*, *B. typhosus*. (*See also* UR. MYCIOPULMONARY SPIROCHÆTOSIS, p. 282).

(The bacteriology of the respiratory tract is at present uncertain.)

Morbid Anatomy.—Mucous membrane of trachea and bronchi red, congested, and covered with muco pus.

HISTOLOGY.—Proliferation and desquamation (catarrh) of epithelial cells and of ciliated epithelium. Mucous glands and mucoid cells active. Exudation on surface containing mucus, desquamated cells, and escaping leucocytes. Submucosa œdematous, leucocytic infiltration, vessels dilated, and glands active.

Symptoms.

ONSET.—As in a 'cold.' General malaise. Heaviness in head. Gastric disturbance, and usually constipation. Hoarseness (from laryngitis) common. Pyrexia slight, rarely 101°–103°. Pulse full.

ONSET OF BRONCHIAL SYMPTOMS.—Cough. Tightness and oppression in chest. Dyspnœa on exertion only.

PROGRESS.—Three stages:—

FIRST STAGE.—Cough dry. Expectoration scanty and viscid.

SECOND STAGE.—Cough loose. Expectoration abundant and mucopurulent. Symptoms become easier.

THIRD STAGE.—Cough often paroxysmal. Expectoration purulent. Other symptoms passing away. In stage of convalescence, condition subsides, or may continue for long period.

No hæmoptysis. Rarely streak of blood from pharynx.

Physical Signs.—

RESPIRATION.—Slightly increased.

ON PALPATION.—Bronchial fremitus.

ON AUSCULTATION.—Numerous râles and rhonchi, altering with coughing.

(Examine bases of lungs for bronchopneumonia.)

Course.—

IN HEALTHY ADULTS.—Reaches third stage in one week, and clears in two weeks.

Acute Bronchitis – Course, *continued.*

IN CHILDREN.—Inflammation may extend to bronchioles, whence areas of collapse and bronchopneumonia (physical signs: patchy dullness and bronchial breathing).

IN OLD PEOPLE.—Mucus accumulates at base, with low pneumonia.

Diagnosis.—Rarely difficult.

IN PNEUMONIA.—Dullness and tubular breath-sounds.

IN BRONCHOPNEUMONIA.—Dyspnœa, high temperature; may be signs of consolidation.

Treatment (for adults).—

AT ONSET.—As for a 'cold': sufficient for mild cases. A warm bed and a hot drink, lemonade or whisky. A hot bath. A wrapper round neck and lin. camphoræ et ammon. to chest. Quinine (tinct. quin. ammon. ʒj t.d.s.) occasionally effective. Avoid chills.

If condition is severe :—

GENERAL TREATMENT.—Bed. Room warm. Air moistened by steam kettle. Bowels open: calomel gr. ij–iv and morning saline. Much fluid. Hot drinks at night promote sleep and action of skin.

DRUGS.—Indications vary with stage :—

FIRST STAGE.—Cough dry and useless. Indication for :—

℞ Vin. Ipecac.[1] | Liq. Ammon. Acetatis.[3] ʒj
Spt. Ætheris Nitrosi[2] āā ♏xx | Aq. Camphoræ ad ℥ss
Four-hourly.

[1] Expectorant and laxative; [2], [3] Diaphoretics.

In healthy adults, give vin. antimonialis ♏xx in place of ipecacuanha.

At night.—Hot drinks. Pulv. ipecac. co. gr. x to aid sleep. Paraldehyde ʒj if necessary.

Inhalation.—Tinct. benzoin. co. ʒj in pint of hot water.

If pulse full and rapid.—Tinct. aconiti ♏j hourly for 6 doses (withdraw if pulse weakens).

SECOND STAGE.—Cough loose. Give stimulating expectorants :—

℞ Vin. Ipecac. ♏x | Tinct. Scillæ ♏xv
Ammon. Carb. gr. iv | Infus. Senegæ ad ℥ss
Four-hourly.

Continue inhalations and applications. Avoid opium.

Severe paroxysms of cough: tinct. belladonnæ ♏x, replacing vin. ipecac.

THIRD STAGE.—Cough persistent. Expectoration free. Opium indicated as sedative :—

℞ Tinct. Camph. Co. ♏xxx | Spt. Chloroform. ♏x
Aceti Scillæ ♏xv | Infus. Cascarillæ ad ℥ss
Six-hourly.

Heroin very valuable as linctus, e.g. :—

℞ Terpin Hydrate	gr. j	Alcohol (90 per cent)	♏ix
Acetomorphine HCl	gr. $\frac{1}{20}$	Glycerin	ad ʒj
Menthol	gr. $\frac{1}{24}$		

(*London Hospital Pharmacopœia.*)

Opium is contra-indicated if any cyanosis is present.

CONVALESCENCE.—Tonics.

II. CHRONIC BRONCHITIS.

Etiology.—(1) Following acute bronchitis, repeated attacks. (2) With renal, cardiac, and lung affections; common with gout.

AGE.—In later years.

SEASON AND CLIMATE.—'Winter cough.' Very frequent in Great Britain.

Morbid Anatomy. Mucous membrane of bronchi atrophied and thin.

HISTOLOGY.—Little ciliated epithelium is present: layer of cuboidal cells remaining on basement membrane, or no cells left. A few leucocytes on surface. Fibrosis and some round cells in submucosa. Emphysema present.

Symptoms.—Recurrent winter attacks, or exacerbations. Patient may be free in summer.

1. SHORTNESS OF BREATH, marked on exertion.
2. COUGH.—Especially troublesome at night. Paroxysms may cause giddiness.

SPUTUM.—Usually abundant, mucopurulent, most in morning. Rarely none.

GENERAL HEALTH.—Often good. No fever. Subject frequently become thin, but are often very stout persons, when cough is most trying.

EMPHYSEMA (rarely absent), and renal, cardiac, and other diseases present influence symptoms.

Physical Signs.—Mainly of emphysema: chest distended, expansion slight, prolonged expiration. Numerous rales and rhonchi.

Variations in Type.—

1. DRY CATARRH (bronchitis sicca).—Not uncommon. Sputum scanty; severe and obstinate paroxysms of coughing.
2. BRONCHORRHŒA.—Sputum in large quantities (may be several pints daily). Usually purulent, in others watery (bronchorrhœa serosa). May persist for years, dilatation of tubes commonly occurring.
3. PUTRID BRONCHITIS.—Sputum fœtid: separates into two layers, upper fluid and frothy, and lower thick, containing Dittrich's plugs. Bronchiectasis probably always present.

Treatment.—*Indications*: (1) General treatment important, especially to prevent recurrences; (2) Temporary measures to relieve

Chronic Bronchitis—Treatment, *continued.*

symptoms; (3) Treat associated diseases—gout, heart, etc. Cardiac failure of special importance, owing to 'back-pressure' on lungs.

GENERAL TREATMENT.—Mild climate in winter (South of England, Egypt, Florida, California). Special care during changes of temperature, climate, or residence. Good ventilation and warm fire. Diet rich in fats (cream or cod-liver oil). Breathe through nose, especially at night.

In exacerbations treat as for acute bronchitis.

TREATMENT OF SYMPTOMS.—Moderate cough and free expectoration good for patient. General treatment often relieves symptoms.

For Morning Cough, saline draught:—

℞ Sod. Bicarb.	gr. xv	Spt. Chloroform.	♏ v
Sod. Chlor.	gr. v	Aq. Anisi	ad ℥ j

In equal amount of warm water.

If Cough Distressing, some or all of the following:—

1. Lozenge (Troch. Glycyrrhizæ, *Brompton Hospital*):

℞ Extracti Glycyrrhizæ	gr. iij	Troch. Acaciæ	gr x
Olei Anisi	♏ss		

Occasionally.

2. Linctus:—

℞ Syrupi Scillæ		Syrupi Papaveris	
Syrupi Limonis		Syrupi Tolu	āā ♏xv

Occasionally.

3. Mixture:—

℞ Tinct. Nuc. Vom.	♏v	Spt. Chloroform.	♏v
Ammon. Carb.	gr. iv	Infus. Senegæ	ad ℥ss
Tinct. Scillæ	♏xv		

t.d.s.

If Sputum Viscid, give potassium iodide:

℞ Pot. Iod.		Pot. Bicarb.	gr. xv
Ammon. Carb.	āā ij	Aq. Camphoræ	℥ss

Inhalations useful: tinct. benzoin. co. ʒj to pint of very hot water.

If Cough very Troublesome, give heroin or tinct. camph. co. (*see* Acute Bronchitis).

For Night Cough.—Pil. ipecac. c. scilla (B.P.) gr. iv at night; also heroin.

If Cough is Paroxysmal, add tinct. belladonnæ ♏iij to pot. iodide mixture.

Sleeplessness.—With free expectoration, too long periods of sleep are inadvisable. Insomnia usually controlled by general measures. If necessary, alcohol or paraldehyde (ʒij) at night. Morphia contra-indicated by severe emphysema or cyanosis: best as pulv. ipecac. co. gr. x at bedtime, or as heroin.

III. BRONCHIECTASIS.

(*Dilatation of the Bronchi.*)

Varieties.—

1. CONGENITAL.—Rare, in children; clinically unimportant.
2. TRAUMATIC.—Foreign bodies in bronchi. Aneurysm or neoplasms pressing on bronchi
3. PURE.—
   a. ACUTE.—Rare. May follow whooping-cough.
   b CHRONIC.—Usual form.

ACUTE BRONCHIOLECTASIS in children. Resembles suppurative bronchopneumonia.

Mode of Production.—Doubtful Essential factor is *weakening of bronchial walls* Of importance therefore are factors (1) tending to weaken walls directly, and (2) leading to retention of secretion, with subsequent reaction on walls by distention and inflammation. Such factors are: (1) Direct inflammation of bronchi, chronic bronchitis, foreign bodies, (2) Pulmonary fibrosis, pleuritic adhesions, pressure of neoplasms and aneurysms, preventing expulsion of secretion

Morbid Anatomy.—Lower lobe more affected than upper. Dilatations are *cylindrical* or *saccular*, both forms may co-exist; the latter produces symptoms. Wall of saccules smooth (contrast to rough tuberculous cavities), at bases of lungs some ulceration. Contents fœtid Lung tissue cirrhotic. Commonly, but not invariably, pleuritic adhesions and pleurisy

HISTOLOGY.—Dilatations lined with flattened epithelium Muscular layer atrophied Peribronchial and general fibrosis.

Symptoms.—Usually distinctive and diagnostic

1. COUGH.—*In paroxysms.* Especially in morning Often one or two daily May follow change of posture (secretion irritating normal tube).
2. FŒTID SPUTUM.—(*a*) Large quantities; (*b*) *Sweet, very offensive odour* Separates into three layers: (i) Froth; (ii) Fluid; (iii) Heavy deposit, containing Dittrich's plugs, leucocytes, and crystals. Occasionally absent in early stages.

HÆMOPTYSIS.—Rarely large, but frequent in small amounts.

CLUBBING OF FINGERS.—Very common

GENERAL CONDITION.—Pallor, some cyanosis, but general health often fair. Pyrexia slight or nil.

Physical Signs.—Generally unilateral. Chronic bronchitis and emphysema often present.

INSPECTION.—Chest may show contraction and displacement of organs due to fibrosis of lung.

PERCUSSION.—Impaired, not absolute, dullness.

AUSCULTATION.—Signs change rapidly in cavities filling and emptying. If empty, extreme amphoric breathing, râles, and rhonchi. If full, breath-sounds diminished, râles slight.

Bronchiectasis, *continued.*

Complications and Sequelæ.—

1. SEPSIS.—Especially abscess of brain. Septic bronchopneumonia, pleurisy, pericarditis, and gangrene of lung also occur: all fatal.
2. RECURRENT ATTACKS OF BRONCHITIS.
3. ARTHRITIS.
4. HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY. —All stages, from clubbing of fingers—very frequent—to rare typical condition.

Diagnosis.—Usually simple by symptoms Physical signs confirmatory. Cavity at base with upper lobes clear suggests bronchiectasis. Diagnosis from :—

GANGRENE OF LUNG.—Constitutional signs greater.

RUPTURE OF EMPYEMA.—Acute history. Sputum foul but syt fœtid.

TUBERCULOSIS.—In bronchiectasis of upper lobe, tuberculosis excluded by absence of tubercle bacilli.

Prognosis.—In fully developed disease, prognosis very bad. Occasionally fair health for years. Sepsis, cardiac failure, abscess of brain, Gangrene of lung, and rarely hæmoptysis, cause termination.

Treatment.—1. Cure is impossible, but symptoms can be ameliorated.

℞ Extract (1) To promote emptying of cavities; (2) To modify fœtid nature of contents by antiseptics.

Olei Aurantii ℳ x.

2. POSTURAL DRAINAGE OF CAVITIES.—'Postural coughing': head hangs over edge of bed, nearly to floor. Emetics aid children.

3. ANTISEPTICS.

℞ CREASOTE CHAMBER.—Closed room Crude creasote evaporated by lamp. Eyes covered by goggles strapped on. Ears and nose plugged. Duration 15 minutes, alternate days. Watch pulse. Very valuable.

INTERNAL.—Creasote capsules, ℳ ij t.d.s.

INHALATIONS.—For example :—

℞ Iodoformi	gr. j	Chloroformi	ℳ ij
Olei Eucalypti	ℳ x	Spt. Rectificati	ℳ x

10–15 drops inhaled on a respirator

(*City of London Chest Hospital*)

OPERATION.—(Before operation, give above measures long trial). *Remove ribs freely* and drain cavity. Drainage alone fails, as cavities cannot close. Results occasionally satisfactory, with selected cases.

IV. BRONCHIAL ASTHMA.

(*Spasmodic Asthma.*)

Attacks of paroxysmal dyspnœa, principally expiratory, due to spasm of bronchial muscles and oversecretion of mucus. Renal and cardiac asthma is not here referred to.

Protein Hypersensitiveness.—It is now agreed that the essential factor in many cases is hypersensitiveness to a foreign protein; but note: (1) Evidence, at present, does not prove that all cases

are of this type—e.g., asthma with bronchitis over middle age; (2) An attack in a susceptible person may have other origins—e.g., psychical. In a hypersensitive person, an attack occurs when the protein becomes present in the body, which may occur from inspiration, ingestion, or possibly production in the body. Research work leading to this conclusion was based on: (1) Resemblance of asthmatic attack to anaphylaxis experimentally produced in animals; (2) Liability of asthmatics to anaphylactic phenomena after serum injections. Principal evidence is: (1) Skin reactions identifying a causal protein; (2) Effects of treatment.

SKIN TESTS FOR PROTEIN HYPERSENSITIVENESS.—Extracts of many proteins are now procurable. An extract is placed on the skin, which is lightly scarified. If subject is hypersensitive to such protein, an urticarial wheal forms in about 20 minutes. Tests with many proteins usually necessary. A protein is identified in about 50 per cent of asthmatics.

RELATION TO AGE.—In infancy and childhood, positive results obtained in high percentage: fall with increasing age: after middle age very rare.

MULTIPLE SENSITIVENESS.—Positive reactions often obtained to more than one protein, especially with onset in early life. Hence a positive reaction is not proof that such protein is the cause of an attack. Patient's history is frequently a guide.

PROTEINS PRODUCING HYPERSENSITIVENESS.—Very numerous. Include:—

1. INSPIRED.—(*a*) Pollens of grasses, etc., viz., in hay fever; (*b*) Emanations of horses, birds (feathers), cats, etc.
2. INGESTED.—Commonest are: cereals, especially wheat; eggs; potato; milk; various fish and meat.

The following groups are still doubtful.—

3. BACTERIAL.—Proteins of various bacteria, e.g., staphylococci. Walker believes this is common; others have not confirmed his results.
4. METABOLIC.—Proteins produced or products split off during digestion. Histamine, for example, produces similar anaphylactic phenomena.

OTHER FACTORS INFLUENCING OR PRODUCING ATTACKS.—

1. HEREDITY.—In high percentage: attacks usually commence at early age. The inherited tendency is to hypersensitiveness, and is rarely to same protein in two generations.
2. ACQUIRED HYPERSENSITIVENESS.—Usually in older subjects, e.g., a baker to wheat.
3. OCCUPATION.—Importance depends on above.
4. PSYCHICAL ATTACKS.—An asthmatic *knowingly* sensitive, e.g., to a rose, may develop an attack from an artificial rose.
5. REFLEXES.—Constipation, flatulence, gastro-intestinal disturbances, bronchitis may produce attacks: especially in older subjects, and skin reactions nearly always negative.

Bronchial Asthma—Protein Hypersensitiveness, *continued.*

Relation of such factors and attacks to protein hypersensitiveness unknown. Possibly a separate type. In hay fever also, cauterization of nasal mucous membrane or removal of polypi may prevent attacks.

DISEASES ASSOCIATED WITH PROTEIN HYPERSENSITIVENESS.—Evidence established: hay fever, asthma, urticaria. Will probably include some forms of eczema, certain acute gastro-intestinal disturbances, and various 'trophoneuroses', e.g., angioneurotic œdema, Henoch's purpura; perhaps status lymphaticus. Enthusiasts claim migraine, epilepsy, and most recurrent affections.

Pathogenesis of an Attack of Asthma.—During attack, principal difficulty is expiration. Lungs assume position of forced inspiration, and little air passes in or out in spite of violent efforts.

Condition early recognized as involving: (1) Spasm of muscles of smaller bronchi. (2) Swelling of bronchial mucous membrane. These two factors cause obstruction of bronchioles: air cannot be expelled from alveoli, but is drawn in by more powerful inspiratory muscles until lungs are fully distended. A third factor is (3) Excessive secretion of bronchial mucus. This mucus, being retained, is coagulated by ferment, mucinase, in bronchial mucous membrane: on conclusion of attack is forced along spiral bronchioles and expelled as Curschmann's spirals (Hurst).

Morbid Anatomy.—With recurrent attacks, *emphysema* develops. May be extreme in young persons without associated bronchitis. No other changes post mortem.

Symptoms.—Attacks frequently nocturnal, after a few hours' sleep.

ONSET sudden, or with premonitory symptoms of oppression in chest, paroxysmal sneezing, flatulence, polyuria, nervous depression.

PAROXYSM.—Violent respiratory movements with all accessory muscles; short inspiration, long wheezy expiration; little air entry. Respirations slow. Patient pale or dusky; anxious; cold sweat. Small pulse. At height of distress, paroxysm diminishes. Is never fatal.

CONCLUSION.—Rapid. Great relief. But paroxysm may recur.

COUGH.—Slight until end of paroxysm. Then patient brings up *viscid sputum* (*see below*).

DURATION.—Few minutes to several hours.

Physical Signs in Paroxysm.—

POSITION.—Patient bends forward, grips objects tightly to fix scapulæ. Head thrown back. Shoulders raised: scaleni and sternomastoids contracted to lift thorax.

INSPECTION.—Thorax fixed in complete inspiration. Diaphragm lowered.

PERCUSSION.—Hyper-resonance.
AUSCULTATION.—Numerous râles and noises. No intake of air.

Sputum.—
CURSCHMANN'S SPIRALS.—Expectoration commences as paroxysm passes: at first viscid, then looser. Contains small gelatinous masses, being spirally twisted casts of small bronchi. Microscopically, when unravelled, these consist of a clear central thread with mucin fibrils twisted round; often numerous eosinophils are embedded. Spiral ascribed to rotary action of ciliated epithelium. Spirals almost diagnostic of true asthma, but absent in old cases with emphysema. Very rarely recorded in acute phthisis, but no eosinophils. May continue two or three days after paroxysm.
CHARCOT-LEYDEN CRYSTALS.—Colourless octahedral crystals. In other conditions also. Of no known importance.

Blood.—Marked eosinophilia may be present, these cells forming 5 to 30 per cent, or more, of total leucocytes.

Prognosis.—In children, attacks may cease. With repeated paroxysms emphysema develops. Prognosis depends on this and cardiac condition.

Treatment.—
BETWEEN PAROXYSMS.—
(1) GENERAL TREATMENT.—General health important. Treat constipation, flatulence, etc. For night asthma: light evening meal, avoid fatigue late in day, sleep before dinner.
(2) FOR PROTEIN HYPERSENSITIVENESS.—
a. Inspiratory.—For HAY FEVER, *see* p. 506. Horse, cat, etc: avoid contact.
b. Food Proteins.—Avoidance is necessary: desensitization usually follows prolonged abstinence: attempted immunization by injections of causative protein or increasing amounts by mouth is ineffectual
Non-specific Immunization by Peptone.—Aims at effecting desensitization to any protein by injections of peptone, intramuscular or intravenous. Peptone by mouth (0·5 gr.), one hour before meals, also tried in migraine, angioneurotic œdema, etc. Under trial, but results doubtful.
3. VACCINES.—With chronic bronchitis, especially older subjects, prepare vaccine from predominant organism in sputum.
4. DRUGS.—With chronic bronchitis, give iodide, e.g.:—

℞ Tinct. Lobeliæ Æthereæ	♏ xv	Spt. Ammon. Aromat.	♏ xx
Potassii Iodidi	gr. v	Aq. Camphoræ	ad ℥j

t. d. s.

5. NASAL TREATMENT.—Remove polypi and correct slight abnormalities, but extensive operations are inadvisable. Cauterization often efficacious temporarily.

Bronchial Asthma—Treatment, *continued.*

TREATMENT OF PAROXYSMS.—(a) *Adrenalin* (1–1000 solution), hypodermic injection of ♏ j–ij, immediately attack commences: usually effective: later in attack less effective, needs larger dose, not exceeding ♏ v. Or (b) *Atropine* gr. $\frac{1}{100}$.

INHALATION OF FUMES.—Often relieves partially, but may aggravate bronchitis:—

Pulv. Stramon. Fol. Pulv. Belladon. Fol. Pulv. Hyoscyami Fol. Potassii Nitratis	āā gr. xv.

To be burnt in a saucer.

V. FIBRINOUS BRONCHITIS.

(*Plastic Bronchitis.*)

Essential feature is expectoration of accurate casts of smaller bronchi and bronchioles. Very rare.

Pathogenesis.—Method of formation of casts unknown. Localization to areas of lung is remarkable. In chronic cases, post mortem, emphysema is constant and tuberculosis frequent; nothing characteristic. Skin diseases not infrequently concomitant

Varieties.—(1) Chronic; (2) Acute.

(1) CHRONIC IDIOPATHIC FIBRINOUS BRONCHITIS—Recurrent attacks; similarity of casts shows repeated involvement of same area. Not fatal, except rarely by asphyxia. May be several attacks in 24 hours.

(2) ACUTE FORM.—In fevers, typhoid, pneumonia, etc. Considerable mortality. Casts *in situ* at autopsy.

Fibrinous casts are expectorated rarely in chronic heart disease and pulmonary tuberculosis. Also in diphtheria, and small casts in pneumonia.

Symptoms.—Paroxysms of coughing and dyspnœa, concluded by expectoration of cast.

Physical Signs during Paroxysm.—Area involved indicated by diminished breath-sounds and râles. Flapping of cast said to be audible. Collapse of the lung may occur in affected area.

Character of Casts.—Rolled up when expectorated. On unravelling, perfect casts of bronchi. May be 6 in. long. Consist of mucin.

Treatment.—No treatment prevents recurrence. Acute attacks treated as bronchitis: inhalations of steam and emetics may aid expulsion of casts.

CHAPTER LXXXIII.

DISEASES OF THE LUNGS.

I. PASSIVE CONGESTION OF THE LUNGS.

Occurs in two forms: (1) Mechanical congestion (brown induration); (2) Hypostatic congestion or hypostatic pneumonia (splenization of lung).

Mechanical Passive Congestion.—

CAUSE.—Obstruction to return of blood to heart. Occurs especially in diseases of left heart.

MORBID ANATOMY.—Known as 'brown induration' (or 'heart lungs').

MACROSCOPIC.—Bulky, tough, and œdematous. On section: brown surface, turning red in air.

HISTOLOGY.—(a) Increase of fibrous tissue; (b) Capillaries distended; (c) Blood pigment in alveolar walls; (d) Alveoli contain epithelial cells and altered blood pigment.

SYMPTOMS.—When heart compensation fails: dyspnœa, cough, and expectoration from engorgement of lung. Hæmoptysis not uncommon. Breath-sounds impaired and râles at bases.

TREATMENT.—Directed to cause, as in cardiac failure. Bleeding (20 to 30 ounces) of great value.

Hypostatic Congestion or Hypostatic (Low) Pneumonia.

OCCURRENCE.—In enfeebling conditions, especially in old age.

a. FEVERS, especially typhoid.

b. DEBILITATING STATES, especially of brain—e.g., cerebral apoplexy, coma.

c. ABDOMINAL TUMOURS, ASCITES, by direct pressure.

CONGESTION AND COLLAPSE OF BASES result partly from gravity, partly from weak action of respiratory muscles and heart.

MORBID ANATOMY.—When advanced, condition is known as 'splenization of lung.' Bases, especially posteriorly, dark red, solid, airless, engorged, and pit on pressure; may sink in water; cut surface often resembles spleen, drips blood and serum.

SYMPTOMS.—Indefinite. Dyspnœa and cyanosis usually slight at onset; may become marked.

PHYSICAL SIGNS.—Râles at bases. Also diminished breath-sounds, and, when advanced, feeble bronchial breathing and impaired resonance.

PROGNOSIS.—Serious. Often fatal termination of illness. In causal states examine bases daily.

TREATMENT.—

PROPHYLAXIS important: in old persons, typhoid, etc., move patient at intervals of two hours from one position to another.

INDICATION is to support and stimulate the heart.

Diseases of the Lungs, *continued.*

II. ŒDEMA OF THE LUNGS.

Varieties.—*Serous transudation* from capillaries into alveoli and alveolar wall occurs in two forms: (1) Insidious; (2) Acute.

(1) ŒDEMA OF INSIDIOUS ONSET.—Very frequent, and often terminal in cardiac and arteriosclerotic conditions.

(2) ACUTE ŒDEMA OF THE LUNGS.—Rare. It occurs in:—

a. CARDIAC, MYOCARDIAL, AND CARDIORENAL CONDITIONS (as in the insidious form).—These are the usual causes.

b. DEBILITATING CONDITIONS.—E.g., severe anæmia.

c. PARACENTESIS OF THE PLEURA.—'Albuminous expectoration' in rare cases follows withdrawal of pleural exudate (amount withdrawn probably excessive, with rapid disturbance of pressures).

d. ANGIONEUROTIC ŒDEMA.—Probably local manifestation.

e. ETHER ANÆSTHESIA.

f. IDIOPATHIC

Morbid Anatomy. Lungs pale, semi-solid, sodden, markedly pit on pressure, and on section exude much frothy fluid.

Pathogenesis.—Welch's theory: Relative failure of left ventricle, and blood accumulates in lungs until transudation occurs. Accounts for insidious group and corresponding acute forms. Other types possibly have various causes Idiopathic type is probably pneumonic.

Symptoms in Acute Forms.—*Abrupt onset,* with oppression in chest.

1. DYSPNŒA.—Becoming extreme.
2. COUGH.—Short and frequent.
3. EXPECTORATION.—*Copious*: watery frothy fluid · may be sanious. Occasionally no expectoration; patient drowns rapidly.

Distress, cyanosis, pallor, cold sweat, feeble pulse, develop rapidly.

Physical Signs.—Small bubbling râles. Percussion note resonant, may become dull later.

Prognosis.—Serious. May be fatal in few hours and even minutes. Attacks may occur with, e.g., angina pectoris.

Duration.—When there is recovery, 12 to 24 hours.

Treatment.—

BLEEDING (20 to 30 ounces) essential, to relieve left ventricle.

OXYGEN inhalation.

ATROPINE, gr. $\frac{1}{50}$, hypodermically.

RAPID CARDIAC STIMULANTS.—Caffeine sodium salicylate, inject gr. ii · camphor gr. ij in sterile olive oil ♏x; Hoffmann's anodyne, spiritus ætheris (ʒss).

III. HÆMOPTYSIS.

(*Blood-spitting.*)

Blood from mouth, nose, and pharynx is not regarded as hæmoptysis.

Causes.—The following are the most important:—

FREQUENT CAUSES.—

1. PULMONARY TUBERCULOSIS.—(*a*) Early.—Slight; capillary oozing. (*b*) Late.—Copious; vessels eroded.
2. MITRAL STENOSIS.

OCCASIONAL CAUSES.—

3. CERTAIN LUNG DISEASES.—(*a*) Pneumonia. (*b*) Infarct (occurs with heart disease). (*c*) Neoplasm, bronchiectasis, gangrene, abscess. Very rarely in other lung diseases, e.g., emphysema.
4. ANEURYSM OF AORTA.—(*a*) Sac weeps through eroded bronchi. (*b*) From erosion of lung. (*c*) Rupture of sac—copious.
5. ULCERATION OF LARYNX OR TRACHEA.—Syphilis, neoplasm, tubercle.

UNUSUAL CAUSES.—

6. PURPURA AND BLOOD DISEASES.—Very rare.
7. MALIGNANT SPECIFIC FEVERS.
8. ENDEMIC HÆMOPTYSIS of Japan and China (lung fluke—*Distoma pulmonale westermanni*).

DEMANDING SPECIAL ATTENTION.—

9. IN APPARENTLY HEALTHY PERSONS. *See* p. 140.
10. INJURIES TO CHEST WALL. *See* p. 140.
11. VICARIOUS HÆMORRHAGE.—In suppressed menstruation.

Notes on foregoing List.—

COPIOUS, RAPIDLY FATAL HÆMOPTYSIS confined to: (1) Advanced pulmonary tuberculosis (low case incidence), (2) Aneurysm of aorta; (3) In mitral stenosis it may be pro[illegible], but rarely fatal, and usually beneficial.

PNEUMONIA.—Rusty sputum almost constant in early stage, but the prognosis and the definite symptoms render this of slight importance as cause of hæmoptysis.

VICARIOUS HÆMORRHAGE.—Accepted since the days of Hippocrates; now accused of being due to tuberculosis.

HYSTERICAL DECEPTION AND PURE MALINGERING.—Not uncommon.

Diagnosis of Hæmoptysis from Hæmatemesis.—

Hæmoptysis	*Hæmatemesis*
1. Blood coughed up.	1. Blood vomited up.
2. Blood frothy.	2. Blood still, often dark.
3. Reaction alkaline.	3. Reaction usually acid (gastric juice); may be alkaline.
4. Sputum stained for several days.	4. No staining of sputum.

Hæmoptysis—Diagnosis, *continued.*

Patient's opinion usually reliable as to coughing or spitting.

Other points are: Previous history of cough or dyspepsia; tarry stools; tubercle bacilli in sputum; physical examination.

Treatment.—Noticeable are patient's mental excitement and often troublesome cough, which promotes further bleeding.

VARIETIES OF HÆMOPTYSIS.—Slighter forms—e.g., early tuberculosis—need no urgent treatment: importance is in diagnosis. Copious degree in mitral stenosis usually beneficial.

INDICATIONS.—(1) Calm patient; (2) Reduce heart-beats; (3) Prevent flooding of other bronchi.

CONTRA-INDICATED.—Alcohol and stimulants (fainting promotes clotting).

IMMEDIATE TREATMENT.—Reassure patient. Examination brief. Inject morphia gr. ¼ to ½ (calms patient, eases cough, quiets heart). Posture: recumbent, shoulders raised, leaning on elbow on affected side, head hanging down; promotes expectoration and protects unaffected bronchi. Ice to suck.

SUBSEQUENT TREATMENT.—Rest. Light diet. No alcohol. Open bowels with salines.

LOWERING OF PULMONARY BLOOD-PRESSURE. Pulmonary circulation little understood, and attempts to influence it are best avoided. Ergot increases pressure, and is contra-indicated. Tinct. aconiti lowers pressure but weakens heart. Amyl nitrite excites heart. Ipecacuanha may cause vomiting.

DRUGS TO PROMOTE CLOTTING.—No evidence of any value in, e.g., adrenalin, calcium lactate, turpentine, gallic acid, aromatic sulphuric acid. Last is useful as placebo (♏x, t.d.s.).

IV. INFARCT OF THE LUNG.

(*Pulmonary Apoplexy. Hæmorrhagic Infarct.*)

Effusion of blood into air-cells and interstitial tissue. Most common in chronic heart disease and infective endocarditis.

Morbid Anatomy.—Mainly on periphery of lung; circular; dark, firm, raised above surface. Wedge-shaped on section, greatest breadth on surface of lung. Size: Walnut to orange or larger. Often multiple.

RECENT INFARCT.—Dark, solid, resembles blood-clot; pleurisy usual.

OLD INFARCT.—Organization, fibrosis, and contraction occur. If septic embolus (infective endocarditis), rarely may suppurate.

HISTOLOGY.—Blood in air-spaces and walls, but tissue is not destroyed.

Mode of Formation.—Not fully known. Two theories:—

(1) EMBOLUS OF PULMONARY ARTERY.—Embolus from right auricle or systemic veins blocks pulmonary artery; wedge

shape due to distribution of arterioles. Embolus often found, but not always. Latter cases might be thrombosis. Opposed to theory: experimentally blockage of pulmonary artery often fails to produce infarct, blood being supplied by bronchial arteries; also there is frequently absence of disease in sites which could produce pulmonary embolus.

(2) DIAPEDESIS FROM DISTENDED VESSELS (Hamilton).—Due to obstruction to and alteration of pressure in pulmonary circulation. Wedge shape then explained by arrangement of terminal bronchi.

Symptoms.—Indefinite With chronic heart lesions, diagnosed by sudden onset of: (1) Pain in side; (2) Dyspnœa; (3) Cough; (4) Blood-stained sputum.

Physical Signs.—Pleural friction. If large, impaired resonance, tubular breathing.

Embolism of Pulmonary Artery or large branches commonly results from detachment of venous thrombi—e.g., from femoral vein. Dyspnœa and cyanosis extreme, onset rapid, and mortality high.

V. COLLAPSE OF THE LUNGS.

The fœtal lung may fail to expand after birth, constituting *congenital atelectasis*; lungs airless, pale, general resemblance to liver tissue. Of no clinical importance.

Collapse during life may occur in two forms: (1) Lobular; (2) Massive collapse—also known as 'lobar' collapse.

1. LOBULAR COLLAPSE.

Small scattered areas of collapse, e.g., in bronchopneumonia, are very common.

Occurrence.—Definite pulmonary disease always present ..., bronchopneumonia and capillary bronchitis, especially in children; bronchiectasis; chronic bronchitis; œdema of bases, especially in old people, and in debility—e.g., enteric; occasionally in whooping-cough; rarely, fibrinous bronchitis.

Morbid Anatomy.—Collapsed lobular areas are depressed below general surface, of purple tint, definite margin, and firm to pressure. On section: airless, fluid scanty, sinks in water. Especially in lower lobes and at margins. Areas may be extensive and lobar.

Symptoms and Signs.—Dominated by associated conditions. Increase of dyspnœa and cyanosis and rapid pulse occur. Physical signs usually indefinite: in children, inspiratory retraction of lower costal spaces and abdomen.

Large Areas of Collapse.—Occur as mechanical result of pressure, e.g., pleural and pericardial effusions, aneurysm, neoplasm, and (less extensively) enlarged heart: also in pneumothorax (special physical signs).

Lobular Collapse of the Lungs—Large Areas, *continued.*

PHYSICAL SIGNS.—Percussion note dull, air entry slight, breath-sounds definitely or distantly tubular or diminished, or even entirely absent, adventitious sounds absent. May closely resemble and is often mistaken for pneumonia or pleural effusion (and often repeatedly tapped).

2. MASSIVE COLLAPSE.*

Acute collapse of an entire or a large portion of a lung.

Occurrence.—

1. POST-OPERATIVE COLLAPSE.—Especially, but not confined to, abdominal operations near the diaphragm.
2. PARALYSIS OF MUSCLES OF RESPIRATION, e.g., diphtheritic.
3. INHIBITION OF MUSCLES OF RESPIRATION, e.g., pneumonia.
4. TRAUMA.—Usually, but not invariably, to the chest wall.
5. BLOCKAGE OF A LARGE BRONCHUS, i.e., by a foreign body.

Mechanism of Massive Collapse.—In civil practice principally studied in post-operative cases. During the war occurred frequently, most instances falling into groups: (1) *Penetrating wounds*, i.e., with hæmothorax: (*a*) Homolateral; (*b*) Contralateral, viz., on opposite side to injury. (2) *Non-penetrating wounds*: (*a*) Homolateral; (*b*) Contralateral. No satisfactory explanation of last exists. There are two rival theories of the mechanism of collapse:—

(1) OCCLUSION OF AIR-PASSAGES.—When healthy lungs are removed from the body at autopsy, complete collapse does not occur: the collapse of the bronchioles rapidly occludes the lumen, and hence air in the alveoli cannot escape. But in life, such imprisoned air is absorbed by the blood, and hence complete collapse in the affected area follows occlusion of a bronchiole or bronchus. Collapse, lobular or lobar, hence results from any cause of such an occlusion. Is undoubted cause of massive collapse in blockage of a bronchus. Elliott and Dingle (*Lancet*, 1914) found: (i) No paralysis of diaphragm is present in post-operative collapse; (ii) Paralysis of diaphragm in cats is not followed by collapse of lungs; they also believed that (iii) Profuse bronchial secretion is present in all cases of post-operative collapse, and produces it by blocking of bronchioles; and did not consider inactivity of muscles of respiration to be a cause of collapse.

(2) INACTIVITY OF MUSCLES OF RESPIRATION.—May result from: (*a*) Paralysis—e.g., diphtheritic, myasthenia gravis; (*b*) Inhibition, e.g., post-operative, trauma, pneumonia (very rare). The following points may be noted:—

(1) Bronchitis of all types is extremely common, but never produces massive collapse. Hence another factor must be present.

*Quart. Jour. of Med. vols. xii. and xiii. *Lancet*, 1914. *Brit. Med. Jour.* 1914.

(ii) Chest wall is always immobile and retracted, and, together with the diaphragm, in position of full expiration. (Confirmed in numerous war cases: but diaphragm found acting by Elliott.)

(iii) In many war cases there was no bronchitis or any pulmonary condition which could block a bronchus. Also in some cases in civil practice.

CONCLUSION.—Immobility of chest wall is primary factor in production of massive collapse.

Probable mode of action: Owing to chest wall being fixed in position of expiration, air entry is slight; air present in alveoli is then absorbed by the blood and not replaced; the lung consequently collapses, and hence the lumen of the bronchioles becomes occluded; thus collapse proceeds rapidly and massively.

Presence of bronchitis will aid such process. (Factors of intra-thoracic pressure, elasticity of lung, etc., are very complex, and their influence in massive collapse has not been estimated.)

Symptoms.—Vary with cause, but often slight at complete rest. Exertion, even moderate, produces marked dyspnœa, rapid respiration and pulse, and sometimes cyanosis and distress. Cough often slight: may be no sputum.

Physical Signs.—(1) Chest wall immobile and retracted—smaller than unaffected side; (2) Heart displaced towards affected side; (3) At affected base, as described above for large areas of collapse. *Fluid in the pleura* necessarily alters these signs, but with collapse a heart in its *normal position* is consistent with a considerable amount of fluid.

X RAYS.—Often diagnostic.

Progress.—Heart usually returns to normal site in about three weeks: often longer: occasionally in ten days. As lung expands, râles and sputum are common. Pneumonia, pleurisy, and effusions may develop.

Treatment of Extensive Collapse.—Varies with cause. General indications are to maintain strength with alcohol and stimulants, to provide oxygen for the tissues by oxygen inhalations or artificial respiration (in certain cases), and to promote respiration and expectoration.

VI. CHRONIC INTERSTITIAL PNEUMONIA.

(*Cirrhosis of the Lung. Fibroid Phthisis.*)

Fibrosis of the lung occurs in various conditions, especially tuberculosis. In many forms pathology doubtful and exclusion of tuberculosis difficult. Clark, Hadley, and Chaplin classify fibroid disease of the lung as: (*a*) Pure fibroid—no tuberculosis; (*b*) Tuberculo-fibroid—tuberculosis with fibroid course; (*c*) Fibro-tuberculous—primarily fibroid, becoming tuberculous.

Chronic Interstitial Pneumonia, *continued.*

Fibrosis may be: (1) Local—portion of lung; (2) Diffuse—involving one or both lungs.

1. LOCAL.—Occurs in:—
   TUBERCULOSIS: a constant change.
   NEOPLASMS, ANEURYSM compressing bronchi.
   INFARCTS.
2. DIFFUSE—Occurs in:—
   CHRONIC TUBERCULOSIS.—Fibroid phthisis. Unilateral.
   ACUTE PNEUMONIA Very rare sequel; resolution fails, plugs organize, alveolar walls thicken (gray induration). Massive lobar type.
   BRONCHOPNEUMONIA—May occur in measles, whooping-cough, influenza, recurrent bronchopneumonia, and bronchitis Fibrosis extends from bronchi. Bronchi dilated or bronchiectasis present. Insular type.
   PLEUROGENOUS INTERSTITIAL PNEUMONIA—Pleura thickened and fibrotic process spreads into lung in strands. Deeper areas of lung unaffected.
   PNEUMONOKONIOSIS (*see* p 531)—From inhalation.
   SYPHILIS (*see* p 269).

Origin.—Fibrous process may commence in and spread from: (1) Peribronchial tissue, as in bronchopneumonic form; (2) Alveolar wall, as in pneumonic form; (3) Pleura and interlobular septa, as in pleurogenous form.

Morbid Anatomy.—Two main types: (1) Massive or lobar; one of more lobes affected. (2) Insular or bronchopneumonic; scattered areas

1. MASSIVE TYPE—Unilateral, usually lower lobe. Thorax and organs affected by contraction of lung.
   LUNG.—Small, gray, airless, tough. *Pleuritic adhesions* constant. Bronchial dilatations not uncommon. If tuberculous: cavities at apex frequent, and other lung tuberculous In pleurogenous form, pleura often half an inch thick Unaffected lung emphysematous.
2. INSULAR OR BRONCHOPNEUMONIC TYPE,—Scattered pigmented fibroid areas; especially lower lobe; often central intervening tissue emphysematous. Pleura little affected Bronchial dilatation and bronchiectasis very frequent Most common type of non-tuberculous fibrosis.

'RETICULAR' FORM.—Intersecting fibrous strands Very rare. Hypertrophy of the heart common.

Symptoms.—Condition chronic. Light work possible for many years. Symptoms of chronic bronchitis with exacerbations: (1) Chronic cough with expectoration; (2) Shortness of breath, often only on exertion. If bronchiectasis present, sputum fœtid and other signs. *Pyrexia*: often absent, when chronic. *With cardiac failure*, usual symptoms.

Physical Signs.—*Inspection* of main importance, results produced by contraction of fibrosed lung.

INSPECTION –

(1) Chest wall on affected side retracted and shrunken, shoulder drawn down, shoulder muscles wasted. Respiratory movement slight.

2. Heart displaced to affected side, may be entirely on right if to left, large area of pulsation, and apex beat displaced upwards and outwards.

3. On measurement, affected smaller than unaffected side.

PALPATION. Tactile fremitus *usually* diminished.

PERCUSSION. Varies with dilatation of bronchi, bronchiectasis, and cavities. In general, resonance diminished.

AUSCULTATION. Also varies as for percussion. In general, breath sounds feeble at base, with bubbly rales, at apex, often amphoric quality.

UNAFFECTED SIDE. Emphysematous, bulky and hyperresonant.

All grades of above description occur.

Sputum.—Examine for tubercle bacilli. Secondary infection common in all types.

Diagnosis. *Inspection usually sufficient.*

DISTINCTION OF TUBERCULOUS FROM OTHER TYPES—(1) Tubercle bacilli in sputum (may be absent). (2) Opposite lung usually shows signs at apex. Often impossible to distinguish.

PRESENCE OF BRONCHIECTASIS. Sputum fœtid.

Prognosis. Fair in absence of bronchiectasis and sepsis. Often 15 to 20 years. Death from failure of right heart, rarely hæmorrhage, amyloid disease, gangrene of lung.

Treatment. Mild climate and general careful life. Treat as for chronic bronchitis and bronchiectasis, according to symptoms.

VII. PNEUMONOKONIOSIS

Definition.—Fibrosis of lung due to inhalation of dust in various occupations. The various forms include (1) Anthracosis (coal miner's disease), (2) Silicosis due to mineral dust, as in steel grinder's phthisis or 'grinder's rot' and gold miner's phthisis on the Rand, (3) Siderosis, from metallic dust.

Fate of Inspired Particles. The air passages can dispose of large amounts of dust, the nose and pharynx arresting some.

IN TRACHEA AND LARGE BRONCHI. *Mucous corpuscles* ingest particles, cilia sweep them along, cough finally ejects them in sputum. With bronchitis, polynuclear neutrophils also present.

IN SMALL BRONCHI.—*Alveolar cells* desquamated from air cells ingest particles.

ALVEOLI – Little or no dust reaches these.

WHEN DUST IS EXCESSIVE, some particles penetrate bronchial mucosa, reach lymph spaces, and are ingested by phagocytic connective-tissue cells. Yet lungs permanently black with carbon may crepitate throughout. Finally, irritation of particles causes a spreading interstitial sclerosis.

Pneumonokoniosis, *continued.*

Morbid Anatomy.—Particles, after passage through mucosa, are arrested in :—

1. TRACHEAL AND BRONCHIAL GLANDS.—These become sclerosed and hard. From periadenitis often adhere to pulmonary veins. Rarely, particles enter circulation by this route and reach liver and spleen.
2. PERIBRONCHIAL AND PERIARTERIAL LYMPH NODES. Fibrosis commences here and spreads into lung tissue, forming *fibroid nodes*, which by coalescence may involve large areas.

In addition to *fibrosis*, other changes are :—

CHRONIC BRONCHITIS.—Constant, and cause of symptoms.
EMPHYSEMA, of unaffected portions.
SOFTENING OF FIBROID AREAS, often with formation of cavities, not uncommon.

MACROSCOPIC APPEARANCE OF LUNGS depends on above changes, varying somewhat with cause, and with presence of tuberculosis.

IN ANTHRACOSIS : Lungs black ; pleural adhesions ; pleura thickened, with extensions into lung ; lung tissue hard and airless ; on pressure, cut surface exudes black fluid ; areas of emphysema usual, mainly marginal. May be scattered, stony hard portions (lung stones). *Bronchial glands* enlarged, black, and often adherent.

Occurrence of Tuberculosis.

ANTHRACOSIS.—Tuberculosis uncommon. Death-rate among coal-miners lower than normal population.

SILICOSIS AND OTHER FORMS.—Tuberculosis very common.

Mode of Entry of Particles.—In some experiments, particles introduced into stomach have reached lung and produced pigmentation and fibrosis. Inhalation generally accepted as usual mode of entry.

Occurrence of Pneumonokoniosis.

METALLIFEROUS MINES.—In dry and dusty mines incidence very high. In dry rock-drilling, great mortality. Tuberculosis and pneumonia frequent complications. Common in Cornish tin and South African gold mines.

STEEL-GRINDING.—'Grinder's rot' in Sheffield.

CHINA AND EARTHENWARE TRADES. Special incidence among 'scourers' cleaning sand off porcelain after removal from kiln.

COTTON WORKERS.

In these and similar trades mortality now greatly reduced by working over gratings with down-drafts, by wet methods, by screens, by respirators, and by washing hands before eating.

Symptoms.—Several years elapse before onset of symptoms.

1. DYSPNŒA.—Marked, and out of proportion to physical signs ; probably from emphysema.
2. COUGH.

3. SPUTUM.—Often characteristic—e.g., 'black spit' of coal-miners, and gritty in silicosis.
Symptoms of tuberculosis when secondary infection occurs.

Physical Signs.—Very various, but not distinctive. Depend on chronic bronchitis, emphysema, and cavitation.

Diagnosis.—By occupation and symptoms. *Examine sputum* for tubercle bacilli.

Treatment.—As for chronic bronchitis and emphysema.
PROPHYLAXIS in the mines and workshops is of great importance

VIII. EMPHYSEMA.

Definition.—A disease of the lungs characterized pathologically by dilatation of the alveoli and atrophy of the alveolar walls.

Types of Emphysema.—(1), Hypertrophic; (2) Atrophic; (3) Compensatory; (4) Acute vesicular; and (5) Interstitial. Hypertrophic emphysema is of principal importance. The other types are briefly referred to at the end of the section.

1. HYPERTROPHIC EMPHYSEMA.

Also known as idiopathic or Jenner's large-lunged emphysema Characterized by enlargement of the lungs, dyspnœa, and cyanosis.

Etiology.—
DILATATION OF ALVEOLI is the primary change. Theories of origin:—

1. INSPIRATORY PRESSURE (Laennec)—Forcible inspiration distending the alveoli. Can explain compensatory emphysema and possibly type following asthma, but not accepted general cause.
2. EXPIRATORY PRESSURE (Jenner). On forcible expiration, e.g., cough, glottis is closed and thorax compressed, over-distention of alveoli results, firstly at apex and anterior margins of lungs, these being least protected. Theory supported by occurrence in players of wind instruments.
3. CONGENITAL WEAKNESS OF LUNG ELASTIC TISSUE.—Family tendency to emphysema recognizable.
4. FREUND'S THEORY.—Ascribes primary change to ossification of costal cartilages, emphysema being secondary. Not accepted.

Expiratory theory, possibly with some congenital weakness, is accepted as main cause.

INFLUENCE OF OCCUPATION.—Important factor. Frequent in those exposed to strain of lifting weights, or local strain as in players of wind instruments and glass-blowers.

INFLUENCE OF COUGH.—Emphysema almost constant with chronic bronchitis. May follow whooping-cough.

SPASMODIC ASTHMA.—May produce in childhood pure emphysema without bronchitis.

Hypertrophic Emphysema —Etiology, *continued*

AGE —Common in middle and late life. No age exempt occurs in children from asthma whooping cough, and recurrent bronchitis

SEX —Commoner in males

GOUT AND GRANULAR KIDNEY not uncommon concomitants

Pathology.— The sequence of events is briefly as follows The air cells distend from the over-pressure This distention stretches alveolar walls and squeezes capillaries and also possibly over stretches elastic tissue Malnutrition from lack of blood leads to atrophy of alveolar walls and finally to rupture spaces resulting composed of several air spaces By coalescence of areas definite bullæ may form Microscopic appearances correspond large air spaces lined with pavement epithelium, thin walls little elastic tissue and diminished capillaries Emphysema is thus established in lungs with diminution of alveoli and capillaries which perform aeration of blood and of elastic tissue which contracts lung Two sequelæ follow

1. Expiration becomes prolonged The loss of elastic tissue diminishes power of contraction increased duration is a partial compensation
2. Inspiration becomes excessive in attempt to compensate the deficient oxygenation of blood which results from fewer alveoli and capillaries

With excessive inspiration and deficient elastic recoil and expiration lung permanently assumes condition of full inspiration Subsequently (1) Chest wall becomes fixed in full inspiration with ossification of costal cartilages (2) Diaphragm is depressed In this condition inspiration is effected by accessory muscles of respiration, scalenes and sternomastoids which lift entire thorax

With reduced capillaries and deficient oxygenation work of heart is increased, right heart hypertrophies and dilates Atheroma of pulmonary artery not uncommon Finally cardiac failure occurs

Morbid Anatomy. —

THORAX —Barrel shaped Costal cartilages calcified

ON REMOVING STERNUM Lungs do not collapse Anterior margins occupy anterior mediastinum and cover heart

LUNGS ON REMOVAL Do not collapse Bulky pale and pit on pressure, and characterized by soft downy feel Apex and anterior margins most affected, may be large bullæ Changes bilateral Bases often congested and œdematous

BRONCHI In large tubes, chronic bronchitis In small tubes, some dilatation but bronchiectasis not common

HEART —Hypertrophy and dilatation of right ventricle Often atheroma or dilatation of pulmonary artery

OTHER ORGANS —Effects of venous congestion

Symptoms —Result from deficient oxygenation of blood *Chronic bronchitis* is invariably present except in children with spasmodic asthma.

1. DYSPNŒA.—Constant, especially on exertion. Paroxysmal attacks may occur.
2. CYANOSIS.—Extreme grade may occur with fair health.
3. COUGH FROM CHRONIC BRONCHITIS.—Rarely absent. Sputum usually scanty.

With age and recurrent bronchitis condition advances Obesity not infrequent, but wasting in some cases. In children, dyspnœa on exertion may be sole symptom.

Physical Signs.—Bilateral.

INSPECTION.—Thorax 'barrel-shaped'; anterior-posterior diameter increased. Position of full inspiration: shoulders raised, clavicles prominent, intercostal spaces wide, sternal angle increased. Apex beat not visible. May be epigastric pulsation (right ventricle); also inspiratory retraction. Cervical veins prominent. Posteriorly: back rounded and scapulæ almost horizontal.

PALPATION.—Apex beat not palpable Vocal fremitus normal or slightly diminished.

PERCUSSION.—Hyper-resonant. Cardiac dullness diminished or absent.

AUSCULTATION.—*Expiration prolonged*; inspiration short; no interval at end of inspiration Râles and rhonchi. Breath-sounds diminished. Heart-sounds feeble but clear.

Course.—Progressive. Symptoms greatly depend on recurring bronchitis. Thus subject is often fit in summer and an invalid in winter. Care and good climate ward off many attacks, and duration may be 15 or 20 years Finally cardiac failure, or occasionally pneumonia

Prognosis.—Depends on degree of emphysema and bronchitis, and condition of heart and kidneys

Treatment.—The process of emphysema is unaffected by any treatment. *Indications*: (1) Treat or prevent attacks of bronchitis or of asthma (*see* CHRONIC BRONCHITIS and ASTHMA); (2) Alleviate symptoms In addition to treatment for bronchitis and asthma:—

OCCUPATION. Alter if predisposing. Measures are frequently hindered by social position of patient

CLIMATE.—Low altitudes (near sea level), warm, moist, free from dust and wind. High altitudes very unsuitable (rarefied air). Best localities are Assouan and South California; South Coast from Bournemouth to Penzance; Madeira; Algiers.

GASTRO-INTESTINAL MEASURES.—Flatulence aggravates dyspnœa.

a. Blue pill, with morning saline purge, twice weekly, to regulate bowels.

b. Treat flatulence with alkalis and gentian.

EXTREME DYSPNŒA AND CYANOSIS AND CARDIAC FAILURE.—*Venesection* (20 to 30 ounces). Oxygen inhalations. Cardiac stimulants and treatment as in cardiac failure.

COMPRESSED-AIR CHAMBER.—Pressure of 1½ to 2 atmospheres for 1 hour. Relief is transient; must be repeated frequently. Freund's operation, resection of costal cartilages, is of no value.

2. OTHER TYPES OF EMPHYSEMA.

Atrophic Emphysema.—Also known as senile atrophy of the lungs and Jenner's small-lunged emphysema. Is a primary atrophy occurring in old age together with general atrophy; found in old withered people. Condition contrasts with hypertrophic type. Thorax small; ribs oblique. On removal, lungs not bulky, collapse readily; on section, large vesicles recognizable.

Compensatory Emphysema.—Is secondary to pulmonary lesions. Over-expansion of lung tissue results as a necessary sequel from contraction or failure to expand of other portions. Occurs locally near patches of bronchopneumonia or tuberculous scars and cavities, or in fibroid phthisis in the entire unaffected lung. In early stages, alveolar walls stretched; later, atrophy and rupture occur, producing true emphysema.

Acute Vesicular Emphysema.—Rapid distention of the lungs may occur with strong inspiratory efforts Has been found in deaths from asphyxia. Also may occur in bronchopneumonia and asthma, and may be produced by pressure on vagi. Lungs hyper-resonant, with râles and prolonged expiration. Return to normal during life has been observed.

Interstitial Emphysema. Escape of air into subpleural and connective tissue of lungs. No connection with true emphysema. Results from wounds of lungs, rarely from rupture of air-vesicles during violent cough, and occasionally after tracheotomy, the air spreading down from wound. Spontaneous pneumothorax may thus arise in healthy persons.

IX. GANGRENE OF THE LUNG.

Etiology.—A rare condition. Results from putrefaction of necrotic areas. Method of production doubtful, but chief role assignable to anaerobic bacilli May occur in variety of conditions:

1. SEPTIC BRONCHOPNEUMONIA. Origin:
   - (a) ASPIRATION PNEUMONIA.—In paralysis and diseases of larynx, wounds of neck, or in insane persons Most frequent cause.
   - (b) PERFORATION OF NEOPLASM OF ŒSOPHAGUS, ETC; PRESSURE OF ANEURYSM OCCLUDING BRONCHUS; RUPTURE OF EMPYEMA, OR OF SUBPHRENIC OR HEPATIC ABSCESS; SUPPURATIVE OTITIS MEDIA
   - (c) CONTENTS OF BRONCHIECTATIC or very rarely of TUBERCULOUS CAVITIES.
2. BRONCHOPNEUMONIA, especially following measles. Rare.
3. LOBAR PNEUMONIA.—Occasionally in diabetes or debility. A classical termination, but extremely rare.
4. EMBOLISM OF PULMONARY ARTERY Usually septic. Rarely in enteric.
5. INJURIES OF LUNG.—E.g., gunshot wounds. Very rare.

PREDISPOSING CAUSES.—Diabetes, debility, and possibly alcoholism.

Morbid Anatomy.—Laennec described two types: (1) Diffuse, involving whole lung. Extremely rare. (2) Circumscribed. Line of demarcation surrounds gangrene; outside is area of congestion, and beyond this area of intense œdema. Gangrenous area at first greenish-brown, then softens, and cavity forms, ragged and offensive.

Symptoms.—Onset usually insidious. Prostration extreme. Fever variable, slight or hectic. Characteristic are: (1) Fœtor of breath; (2) Sputum, same odour. On standing, sputum forms three layers viz., froth, greenish fluid, and greenish deposit, the latter containing elastic tissue, and often lung tissue, but no Dittrich's plugs.

'Latent' cases are discovered at autopsy, especially in diabetes: gangrenous area without opening into bronchus: no fœtor and no sputum.

Complications.—

1. PULMONARY.—(*a*) Bronchitis—invariable, except in latent form; (*b*) Hæmoptysis; (*c*) Pleurisy; (*d*) Pneumothorax—rupture into pleura.
2. ABSCESS OF BRAIN.—Frequent (cf. BRONCHIECTASIS).

Prognosis.—Recovery rare.

Treatment.—Operative, if possible, drain freely. Otherwise, treat as BRONCHIECTASIS (*see* p. 518).

X. ABSCESS OF THE LUNG.

Suppuration in the lung tissue. Often multiple. Always secondary.

Causes.—

1. ASPIRATION PNEUMONIA.—Paralysis and diseases of larynx, wounds of neck; insanity.
2. EXTENSION OF SUPPURATION FROM EXTERNAL SITES.—Rupture of empyema, subdiaphragmatic abscess, hydatid cysts; fractured ribs; rarely perforating wounds.
3. FOREIGN BODIES IN BRONCHI, BRONCHIECTASIS, PERFORATION OF NEOPLASMS.
4. INFECTIVE EMBOLI.—Multiple subpleural abscesses. Localizing symptoms rare.
5. LOBAR PNEUMONIA.—Rare termination.

Symptoms.—Constitutional symptoms marked, and phenomena of sepsis.

PYREXIA, COUGH, DYSPNŒA, PAIN.

SPUTUM.—(1) Offensive, but not extreme sweet fœtor of gangrene and bronchiectasis; (2) Pus and elastic tissue present.

PHYSICAL SIGNS of bronchopneumonia; rarely of cavity.

Complications.—*Pleurisy* (purulent) invariable if abscess reaches surface. Gangrene of lung, pericarditis, hæmoptysis, abscess of brain. When chronic, amyloid disease.

Abscess of the Lung, *continued.*

Diagnosis.—Difficult. Note elastic tissue in sputum. From: (1) Empyema; (2) Bronchiectasis; (3) Gangrene of lung; (4) Putrid bronchitis.

Prognosis.—Grave. Following pneumonia, may recover; following aspiration pneumonia and foreign bodies, mortality very high.

Treatment.—Single abscess: remove ribs and drain; results fair. Chronic abscess: as in bronchiectasis.

XI. NEW GROWTHS IN THE LUNG.

Varieties.—

BENIGN TUMOURS.—Enchondroma, osteoma, etc. Extremely rare.

MALIGNANT TUMOURS.—

PRIMARY.—Rare. Unilateral. Carcinoma most common, rarely sarcoma, usually endothelioma. Metastases uncommon, except in lymphatic glands. Occur at younger age than most neoplasms.

SECONDARY.—Not uncommon. Especially from: (1) Tumours of bone; (2) Chorion-epithelioma. Also from (3) Breast; (4) Alimentary canal; (5) Hypernephroma (invades renal vein); (6) Pancreas; (7) Suprarenal, (8) Thyroid.

HYDATID CYSTS. Not infrequent site.

SYPHILIS.—Very rare.

Symptoms.—Rarely characteristic. Vary with site involved:—

1. PULMONARY AND BRONCHIAL STRUCTURES. (1) Cough: rarely prominent. (2) Dyspnœa: becomes extreme with bronchial or tracheal pressure. (3) Sputum: 'prune-juice' expectoration, from mixture with blood: diagnostic, but rarely present.
2. PLEURA.—Recurrent pleural effusion suggests neoplasm. While fluid is often clear, yet neoplasm is most frequent cause of persistent bloody effusion.
3. MEDIASTINAL GLANDS. May be involved early, causing pressure symptoms of mediastinal tumour, e.g.: (1) Unilateral œdema of thorax and head (may be extreme); (2) Dilated veins; (3) Pains in shoulder and arm; dysphagia; unequal pupils.

Progressive emaciation and anæmia. Pyrexia usually slight. Symptoms as above may all be present.

Physical Signs.—If unilateral, side may be prominent. Signs vary with presence of effusion, consolidation, and excavation. With enlarged mediastinal glands, resembles mediastinal tumour. Supraclavicular glands may be palpable.

Diagnosis.—Usually difficult.

SPECIAL DIAGNOSIS—

1. By X Rays.—From aneurysm.

2. By Sputum.—(a) *Repeated* absence of tubercle bacilli on examination: this excludes tuberculosis. (b) 'Prune-juice expectoration.'
3. By Character of Pleural Effusion.—(a) Recurrent. (b) Sanious. (c) Cytology: absence of pus-cells and small lymphocytes, presence of endothelial cells; presence of neoplastic cells is extremely rare.
4. By Presence of Primary Tumour.
5. By Symptoms.—(a) Progressive; (b) Wasting; (c) Fever absent or slight. May also be: (d) Pressure signs; (e) Supraclavicular glands.

DIAGNOSIS FROM.—
1. Pulmonary Tuberculosis.
2. Aneurysm.
3. Tuberculous and Renal Pleural Effusion.

Treatment.—Palliative. Disease always fatal. Paracentesis when fluid sufficient to produce symptoms.

CHAPTER LXXXIV.

DISEASES OF THE PLEURA.

I. PLEURAL FLUIDS: THEIR EXAMINATION AND CAUSES.

Methods of Examination.—(1) Inspection; (2) Cytology; (3) Bacteriology. Also (4) Chemical

1. INSPECTION. May be: (a) Clear or turbid; (b) Purulent; (c) Hæmorrhagic; (d) Opalescent.

Hæmorrhagic Exudates (not hæmothorax).—Occur in (1) Tuberculosis: rupture of newly-formed vessels in the exudate. (2) Neoplasm of lung: commonest cause. Very rarely in chronic nephritis, cirrhosis of liver, severe fevers.

Any effusion, previously aspirated recently, may be hæmorrhagic from rupture of blood-vessels.

(Aspirating needle may cause bleeding into effusion.)

Opalescent Exudates (*Chylous Effusions*).—Most frequent in parenchymatous nephritis, rarely in neoplasms or after repeated aspirations: due mainly to a lipoid soluble in alcohol but not ether, 'pseudo-chylous fluid.' True chylous fluid very rare: from lesion of thoracic duct or filaria.

Pneumococcal Fluids.—Usually creamy pus with much fibrin.

Fœtid Odour.—Common when in communication with bronchus; also in bronchiectasis, gangrene of lung.

2. CYTOLOGY.—The cells present may be:—

(a) Small Lymphocytes.—In chronic inflammations; almost always tuberculous; fluid commonly sterile.

Pleural Fluids : their Examination and Causes, *continued.*

(b) POLYNUCLEAR NEUTROPHILS.—In acute inflammations due to pyogenic organisms.

c. ENDOTHELIAL CELLS.—Principal cell in transudates: in effusions due to neoplasms, cardiac failure, nephritis, and non-inflammatory conditions. Fluid sterile.

Neoplasm may be suggested by numerous cells in mitosis.

(d) NO CELLS PRESENT. —Not infrequent in transudates.

3. BACTERIOLOGY.—

a. IN PURULENT EXUDATIONS AND FLUIDS CONTAINING POLYNUCLEAR NEUTROPHILS. — Micro-organisms are:

(i) *Pneumococcus*: most commonly: prognosis good.

(ii) *Streptococcus pyogenes*: prognosis less favourable

(iii) Staphylococcus: rare. Rarely: *B. influenzæ*, *B. typhosus*, gonococcus, bacilli of colon group, e g., *B. coli*, Friedländer's bacillus, *B. pyocyaneus*, etc.

b. IN FLUIDS WITH SMALL LYMPHOCYTES —Tubercle bacilli practically never found. Nature confirmed by injections into animals, if necessary.

Principal Causes of Pleural Effusions.—

(1) ACTIVE EFFUSIONS (EXUDATES).—

ACUTE INFLAMMATION.—(i) Lungs and pleura: e.g, pneumonia. (ii) Spread from extrathoracic infections: (*a*) Extension through diaphragm; (*b*) Septicæmia. (iii) Acute rheumatism (never purulent). *Cells*: Polynuclear neutrophils.

CHRONIC INFLAMMATION. —Tuberculosis. *Cells*: Small lymphocytes.

(2) PASSIVE EFFUSIONS (TRANSUDATES). —*Cells*: Endothelial or none.

CARDIAC FAILURE.

ACUTE OR CHRONIC PARENCHYMATOUS NEPHRITIS (may be inflammatory).

CHRONIC INTERSTITIAL NEPHRITIS, and terminal in various debilitating conditions.

INTRATHORACIC NEOPLASMS.

Occasionally with suppuration below the diaphragm.

II. ACUTE PLEURISY.

1. PLASTIC PLEURISY.

(*Fibrinous or 'Dry' Pleurisy.*)

Etiology.—

1. PRIMARY.—Follows cold or chill. In healthy persons rare without effusion.

SYMPTOMS. - (a) Pain in side; (b) Cough; (c) Fever; (d) Friction sound. No fluid present. Symptoms disappear in few days. Frequent cause of pleuritic adhesions. Tuberculosis probably frequently present. (*See* PLEURISY WITH EFFUSION.)

2. SECONDARY TO.—(*a*) Lobar pneumonia. (*b*) Tuberculosis: common initial symptom. (*c*) Various pulmonary diseases when involving pleura: neoplasm, abscess, gangrene, etc.

Clinically, 'dry' pleurisy is frequently early stage of pleurisy with effusion, before occurrence of exudation.

2. PLEURISY WITH EFFUSION.

(*Sero-fibrinous Pleurisy.*)

Etiology.—

1. TUBERCULOSIS.—

(i) *After cold and exposure.* Frequent cause, pleurisy following directly. Most cases are tuberculous; this opinion is based on *frequent* occurrence of following evidence: (*a*) Tuberculous lesions often present, may be latent and previously unsuspected. Lesion sometimes found after aspiration of fluid. Tubercle bacilli in sputum in 15 per cent. (*b*) Tuberculous lesions found post mortem in accidental deaths. (*c*) Effusion cytologically resembles tuberculous fluid (small lymphocytes). (*d*) Effusion injected in large quantities causes tuberculosis in guinea-pigs. (*e*) Tuberculosis subsequently develops; including these with (*a*) accounts for 40 per cent of cases. Evidence is sufficient to prove that many cases are tuberculous; insufficient to show how many—owing to great variation amongst different authorities—or to prove completely that all are tuberculous. Occasionally pneumococci are found, and rarely streptococci, in cases not becoming purulent.

(ii) *Idiopathic.* No cause discernible. Above arguments similarly apply.

2. ACUTE RHEUMATISM. Not uncommon: especially with pericarditis, less often with endocarditis only. Pathology doubtful. May be 'dry' or with effusion.

3. NEOPLASMS OF LUNG.

4. CHRONIC NEPHRITIS, CIRRHOSIS OF LIVER, and DEBILITATING CONDITIONS

5. EXTENSION OF INFLAMMATION.—With inflammation below diaphragm, e.g., subphrenic abscess, or in pericardium, serous effusions may occur.

6. NON-PENETRATING INJURIES TO CHEST.—Less common. Probably tuberculous.

RARE ASSOCIATED DISEASES.—Rheumatoid arthritis, gonococcal rheumatism.

AGE.—None exempt. Most common at 20 to 40 years.

SEX.—Twice as common in males (exposure to cold).

Bacteriology.—Presence of organisms very rare in serous exudations, except in early stages of fluids subsequently becoming purulent.

Morbid Anatomy.—The changes are those common to inflammation of serous membranes. The fluid may be clear or turbid. *Hæmorrhagic exudate* suggests tubercle or neoplasm.

Pleurisy with Effusion -Morbid Anatomy, *continued*

1. CHANGES IN THE PLEURA.--
   MACROSCOPIC. –Early stage: loss of polish, surface injected. Then exudation of fluid or fibrin. Subsequently, fluid may be absorbed and adhesion of injured surfaces occur, or organization of fibrin result in irregular fibrous adhesions and sometimes 'loculated effusions.'
   Adhesions vary from friable bands of lymph to strands of fibrous tissue or to universal adhesion of varying thickness. Adhesions most common near apex, on diaphragmatic surface, and over pericardium.
   HISTOLOGY.—Endothelial cells proliferate and desquamate. Capillaries dilate. Leucocytes, escaping, infiltrate sub-endothelial tissue and reach surface of pleura. Exudation of fibrinous lymph containing endothelial cells and leucocytes
   a. In 'dry' pleurisy, subsequently: Proliferation of connective-tissue cells; processes protruded into lymph, which is absorbed; new blood-vessels form, and fibrous-tissue union of the surfaces follows.
   b. In pleurisy with effusion, subsequently: Fluid is absorbed through veins and lymphatics, and adhesions form as above by organization in the lymph and between injured surfaces.
2. EFFECT OF EFFUSION ON THE LUNG. When effusion small, base and posterior border of lung are collapsed, blue, and airless, but contain blood and œdema. When effusion large, lung is compressed close to the spine, airless, gray, and bloodless ('carnified').
3. DISPLACEMENT OF ORGANS With large effusions the heart and mediastinum are displaced to opposite side, and diaphragm is depressed.

Symptoms of Acute Pleurisy. -

ONSET.—May be :—
1. INSIDIOUS.– Prodromal lassitude and dyspnœa: especially in children and old age.
2. ABRUPT. In children there may be convulsions or vomiting

CHARACTERISTIC SYMPTOMS —(1) Pain in side; (2) Cough, (3) Some dyspnœa; (4) Fever.
1. PAIN IN SIDE.-- Severe, described as 'stabbing'; aggravated by cough, deep inspiration, and sometimes by movement or pressure. *Site*: Usually lower axilla; may be reflected to abdomen, epigastrium, umbilicus, or iliac fossa, thus simulating appendicitis, more frequently in children.
2. COUGH.—Early symptom, occasionally absent; not so severe as pneumonia. Sputum scanty
3. DYSPNŒA.—Slight, from fever and pain; later may be severe if rapid effusion compresses lung. With slow effusion, dyspnœa slight.
4. FEVER.—Rarely exceeds 102° to 103°; rise less abrupt than pneumonia; duration about 7 to 10 days, usually.

POSTURE At onset patient lies on sound side to prevent pressure on inflamed pleura, after effusion, lies on affected side to allow expansion of healthy lung

PNEUMOCOCCAL PLEURISY -Abrupt onset, temperature and crisis may closely resemble acute pneumonia.

Physical Signs of Pleural Effusion.—

CHARACTERISTICS OF EFFUSION -(1) Absence of tactile fremitus, (2) Wooden dullness on percussion, (3) Breath sounds diminished or absent (4) Displacement of apex beat and organs In early stage or 'dry' pleurisy *friction rub* only

INSPECTION -*Displacement of apex beat Immobility of side Occasionally obliteration of intercostal spaces*

PALPATION -*Tactile vocal fremitus absent* or very slight (less definite in children) No œdema of wall Liver and spleen may be depressed

PERCUSSION Characteristic *Absolute wooden dullness* felt by the finger Dullness due partly to fluid and partly to compressed lung earliest at base posteriorly May reach clavicle and include or extend beyond sternum On right merges indistinguishably into liver dullness On left, Traube's semilunar area only obliterated by large effusions Movable dullness is rare, and suggests pneumothorax. Other phenomena observed include

(1) FLETCHER'S SHAPED LINE In the erect posture with medium effusions the upper limit of dullness is not horizontal, but rises from spine to axilla, and then falls to sternum Not marked in large effusions Lying in bed line slopes continuously from spine downwards Is due to position of root of lung, and can be reproduced experimentally

(2) GROCCO'S PARAVERTEBRAL TRIANGLE OF DULLNESS — Triangle of relative dullness along spine on opposite side to effusion apex upwards base ½ to 3 inches Very constant in thin people if fluid reaches 8th dorsal vertebra Also in pneumonia

Theories of Causation: (1) Bulging of mediastinum, (2) Collapse of lung (persists for a time after paracentesis)

3 SKODAIC RESONANCE A tympanitic area often present above limit of dullness Most marked under clavicle with fluid reaching to 4th rib Ascribed to relaxation of lung above the fluid. Resembles tympanitic resonance, with slight impairment of percussion note

AUSCULTATION. —

(1) EARLY STAGE -*Friction rub* (*a*) Usually 'creaking' or 'leathery', with inspiration and expiration, unaffected by cough, disappears with effusion (*b*) Fine crepitations as in pneumonia—less common

(2) WITH EFFUSION —

Breath sounds -(*a*) Over dull area: weak or absent; occasionally bronchial, especially in children (*b*) Above dull area; harsh, loud, and often tubular; may be râles.

Pleural Effusion—Physical Signs, *continued.*

Vocal Resonance.—Usually absent or diminished, rarely bronchophony.

Ægophony.—Nasal twang, common towards upper border of dullness; often at angle of scapula; attributed to thin layer of fluid.

Diminution of breath-sounds depends principally on compression of bronchi, and not on amount of effusion as such, fluid being a good conductor of sound.

EXAMINATION OF HEART.—With displacement, visible impulse is not necessarily true apex. Systolic murmur at base, when much displaced. In left effusions, pleuro-pericardial friction may occur.

MENSURATION.—With large effusions cross-section changes from elliptical to circular. Hence volume increases and size appears larger, with little change in measurement of periphery. Cyrtometer shows shape.

LITTEN'S SIGN.—Movement of the diaphragm. In thin normal persons, supine, with oblique light on the axilla, the 'shadow' of the diaphragm is seen moving with respiration; this is absent in pleural effusion, and often in other pulmonary diseases—e.g., pneumonia. In subphrenic abscess it may be abnormally high.

BLOOD COUNT.—No leucocytosis: count rarely exceeds 12,000 (except in the presence of associated conditions).

RADIOGRAPH.—Fluid gives shadow, often sufficient for diagnosis. In the interscapular region, over site of collapsed lung, tactile fremitus, tubular breathing, and bronchophony may be present even with considerable effusions.

For discussion of pleural effusion without displacement of organs, *see* MASSIVE COLLAPSE OF THE LUNG.

Course.—Variable. Tendency is to be absorbed. Large effusions may compress vessels, causing delay. Aspiration now frequently employed. Immediate prognosis is good.

METHODS OF NATURAL TERMINATION:—

1. ABSORPTION OF EFFUSION.—Following 'chill' and in idiopathic forms, fever subsides by lysis, 7 to 10 days. In type of 'pneumococcal pleurisy,' crisis may occur.
2. LARGE EFFUSIONS ABOVE 4TH RIB.—Absorption slow; often rapid after partial aspiration.
3. EFFUSION PERSISTS UNCHANGED FOR MONTHS.—Especially in tuberculosis.
4. EFFUSION RECURS AFTER ASPIRATION.—Suggests neoplasms. Persistence and recurrence occur: (a) If lung is permanently collapsed and inexpansible—e.g., after carnification; (b) With tight adhesions; (c) With persistent pleural irritation.

ABSORPTION.—Earliest sign: displacement of organs diminishes. Breath-sounds and, later, tactile fremitus return. Rarely redux friction rub. Breath-sounds and percussion note at base may remain impaired: temporarily due to collapse of lung; may become permanent from thickened pleura and adhesions; hence

difficult to certify complete absorption of fluid With rapid absorption, chest wall falls in, and returns but slowly or incompletely, owing to adhesions

ADHESIONS Occur at termination of all pleurisies. may give no physical signs, as after dry pleurisy.

3. EMPYEMA.

(*Purulent Pleurisy*)

Etiology

AGE Commonest under 10 years Then at 20 to 30 years, from incidence of pneumonia

CAUSES

1 SEQUEL OF ACUTE PNEUMONIA -Predominant cause
2 EXTENSION FROM PNEUMOCOCCAL OR SEPTIC FOCI OR SEPTICÆMIA High mortality
3 TUBERCULOSIS
4 TRAUMA Fractured ribs penetrating wounds

Bacteriology PLEURAL FLUIDS) Commonly pneumococcus or streptococcus

Morbid Anatomy.—Inflammation of pleura as in pleurisy with effusion Exudate purulent Opened post mortem, pleuræ usually thickened and often thick pus at base, with clear fluid above

Symptoms.—Characteristics are (1) Symptoms of sepsis viz irregular pyrexia malaise sweating, chills (2) Signs of fluid, (3) Purulent fluid on aspiration (4) Leucocytosis

ONSET Insidious, in course of causal disease In lobar pneumonia temperature does not fall or rises again after a few days

IN CHILDREN Pallor, weakness often vomiting and diarrhœa. Dyspnœa if much fluid otherwise symptoms slight

Physical Signs.—As in pleurisy with effusion Bilateral empyema rare

DIFFERENCES FROM SEROUS EFFUSIONS —(1) Displacement of heart and diaphragm more marked (ascribed to weight of pus), (2) Intercostal spaces may bulge, (3) Œdema of chest wall occasionally, (4) Superficial veins dilated rare

In children loud tubular breathing does not *exclude* empyema.

CLUBBING OF FINGERS Occasionally, in effusions of three or more weeks' duration

LEUCOCYTOSIS —Marked rarely under 15,000

Termination.—

WITH REMOVAL OF PUS After pneumonia prognosis good Occasionally discharge persists owing to (1) Lung unable to expand—e g, after carnification or adhesions, (2) Absence of resolution, and subsequent fibrosis of lung

WITHOUT REMOVAL OF PUS. — Usually no tendency to absorption, and death by exhaustion or perforation

a ABSORPTION - Small effusions Pleura thickens and encloses inspissated pus Very rare.

Empyema—Termination, *continued.*

b. EMPYEMA NECESSITATIS.—Rupture through chest wall: usually anteriorly in 6th space. Prognosis fair. Often chronic discharge.

c. PERFORATION OF LUNG AND EVACUATION.—Usually fatal choking. Pneumothorax may occur.

Perforations into pericardium, stomach, œsophagus, etc., are on record.

Prognosis.—Better in pneumococcal than in streptococcal infections

Complications.—Rare, but commoner than in serous effusions. Pericarditis, pneumothorax, abscess of lung, occasionally abscess of brain, bronchiectasis, gangrene of lung, nephritis.

Clinical Varieties.—

BILATERAL EMPYEMA.—Very rare

'LOCULATED EMPYEMA.'—Pus may be enclosed by adhesions, between lobes, or on surface of diaphragm Physical signs slight, paracentesis difficult.

'PULSATING EMPYEMA.'—Very rare. Effusion large, on left, usually pointing. Pulsation transmitted from heart, probably by pericardial adhesions.

PNEUMOCOCCAL AND STREPTOCOCCAL SEPTICÆMIA—Empyema often overlooked. Condition typhoidal.

TUBERCULOUS EMPYEMA. *See* PULMONARY TUBERCULOSIS.

VARIOUS TYPES OF PLEURISY.

Diaphragmatic Pleurisy.—Inflammation of diaphragmatic pleura. Usually dry; purulent effusions very rare

PAIN.—Over diaphragm and abdomen, or over shoulder.

PHYSICAL SIGNS.—Slight or absent, with marked pain and dyspnœa.

Loculated Pleurisy.—Effusion, usually purulent, separated into loculi by adhesions. Physical signs often doubtful. May be missed on puncture.

Interlobar Pleurisy.—Fluid encysted between lobes. Diagnosis very difficult.

Hæmorrhagic and Chylous Pleurisy.—*See* PLEURAL FLUIDS.

Tuberculous Pleurisy.—*See* PULMONARY TUBERCULOSIS, pp. 147, 165.

DIAGNOSIS OF PLEURISY.

Dry Pleurisy.—*Friction rub* usually distinctive. Diagnosis from (1) Intercostal neuralgia and neuritis no fever; (2) Abdominal conditions; (3) Herpes zoster, before eruption; (4) Pott's disease.

Pleurisy with Effusion.—

METHODS OF DIAGNOSIS.—(1) Symptoms; (2) Signs; (3) Exploratory puncture; (4) X rays. Questions are: (*A*) Is fluid present? (*B*) What is its nature?

A. PRESENCE OF FLUID.—

LARGE EFFUSIONS.—Diagnosis easy: (1) Immobility, (2) Displacement of organs, (3) Tactile fremitus absent, (4) Wooden dullness, (5) Breath-sounds usually absent. *Tactile fremitus* is most reliable of all physical signs.

MODERATE EFFUSIONS, without displacement. Diagnosis from

a *Pneumonia.* In effusions (1) Symptoms not so abrupt, no rusty sputum. (2) Signs: tactile fremitus absent and wooden dullness present.

b Old thickened pleura.

c Neoplasm of lungs.

d Massive pneumonia and collapse of lungs. Rare.

On Left Side.—From pericardial effusions: note area of dullness, no displacement of heart, feeble heart-sounds, marked dyspnœa. Difficulty increased by compression of lung.

On Right Side.—From subphrenic abscess.

B. NATURE OF FLUID. (*a*) Signs of sepsis. (*b*) Withdrawal and examination of fluid.

TREATMENT OF PLEURISY.

Dry Pleurisy. *Indications.* (1) Relieve pain. (2) Prevent extension.

Bed. Open bowels with calomel followed by saline purge.

To relieve pain.—Apply ice bags or strapping (extending over middle line back and front). Hot or cold applications. If severe, inject morphia.

Further treatment depends on cause and on occurrence of effusion.

Pleurisy with Serous Effusion. (Confirmed by hypodermic needle.)

INDICATIONS FOR ASPIRATION.—

1. Fluid increasing, especially when above 4th rib anteriorly.
2. Respiration or pulse affected.
3. Fluid not becoming absorbed (one to two weeks).

CONTRA INDICATIONS TO ASPIRATION.

1. Small effusions causing no embarrassment. Usually become absorbed.
2. Tuberculous effusions. Avoid aspiration if possible. If indications present, remove not more than 20 ounces. (Risk of generalizing tuberculosis.)

PARACENTESIS (ASPIRATION).—Inject novocain freely (½ per cent solution), commencing under skin and then inserting needle down to pleura. No general anæsthetic. Place patient's hand on opposite shoulder. Puncture in 8th space at angle of scapula, at upper border of rib. Small deep incision advisable. Withdraw fluid slowly, not exceeding 50 ounces. Seal puncture with collodion. Strict asepsis.

SYMPTOMS DURING OR SUBSEQUENT TO ASPIRATION.—(1) Coughing: stop aspiration. (2) Faintness, from change of

Pleurisy—Treatment, *continued.*

pressure and shifting of heart: give brandy. (3) Pneumothorax: rare. (4) Subcutaneous emphysema. Very rare: (5) Acute œdema of lungs and albuminous expectoration: fatal. (6) Sudden death: from syncope.

SUBSEQUENT TO ASPIRATION.—Encourage lung expansion by blow-bottles and deep-breathing exercises. Do not strap. Aspiration may be repeated.

AFTER-TREATMENT.—Examine for tuberculosis. Treat all cases with small lymphocytes in fluid as tuberculous. Prognosis fair with treatment.

Empyema.—

1. FROM PYOGENIC ORGANISMS.—

(i) RESECT RIB AND DRAIN FREELY - If amount large, aspirate some 48 hours previously. Patient is never "too ill to stand the operation."

After-treatment. —Drainage tube until no discharge If recovery slow, suction with Sprengel's air-pump assists. Blow-bottles and breathing exercises to assist expansion of lung Fresh air.

(ii) ASPIRATION.—Repeated aspiration may be curative. No objection to its trial with care in selected cases.

(iii) WITH CHRONIC DISCHARGE —Modified Estlander's operation, resection of ribs: allows chest wall to fall in.

2. TUBERCULOUS EMPYEMA.

ASPIRATE PUS. (Resection of rib is nearly always followed by secondary pyogenic infection and chronic suppuration)

III. CHRONIC PLEURISY.

Chronic Pleurisy with Effusion.—Effusion may persist without becoming purulent.

Chronic Dry Pleurisy: Thickened Pleura.—Four varieties:—

(1) SEQUEL OF ORDINARY PLEURAL EFFUSIONS AND EMPYEMA.—Pleuræ very thick. Flattening and lack of expansion at base; impairment of resonance and breath-sounds. Some dragging pain, or no symptoms.

2. PRIMARY DRY PLEURISY.—Commences with acute form, or insidiously. Symptoms slight. Adhesions commonly found post mortem. Litten's sign may be absent. Fibrous tissue, if thick, may invade lung (chronic cirrhosis of the lung).

3. POLYSEROSITIS: POLYORRHOMENITIS.—Very insidious. All serous membranes may be affected. (*See* p. 499.)

4. TUBERCULOSIS OF THE PLEURA.—Caseous masses in pleural membrane. May be bilateral, also in peritoneum, and very rarely in pericardium.

In chronic pleurisy of apex, there may be unilateral sweating of face and dilatation of pupil from involvement of first thoracic ganglion

IV. HYDROTHORAX.

Non inflammatory transudation into pleural cavity (*see* p 539) Presence often suggested by dyspnœa Physical signs as pleural effusion In heart lesions is more frequent on right, possibly from pressure on azygos vein by dilated right auricle Renal effusions are bilateral Character of fluid pale, specific gravity not above 1018, no fibrin, little albumin, cells endothelial or absent, sterile Pleura smooth

Treatment. Aspiration repeated if necessary

V PNEUMOTHORAX:

Hydropneumothorax: Pyopneumothorax

Pneumothorax is air in the pleural cavity Fluid is almost always present i e hydropneumothorax or if purulent, pyopneumothorax Owing to negative intrapleural pressure, when air enters, lung collapses, and mediastinum is displaced to opposite side

Varieties.—There are three forms of pneumothorax —

1 OPEN Perforation patent Pressure is atmospheric
2 CLOSED Perforation sealed
3 VALVULAR —Air enters during inspiration and cannot escape during expiration

In last two forms intrapleural pressure may, and usually does exceed the atmospheric especially as fluid collects hence displacement of organs is extreme

Valvular form is most frequent

(*Note* —Pneumothorax due to gunshot wounds of the chest possesses certain special features and is not considered in this section)

Etiology.—Pulmonary tuberculosis accounts for at least 80 per cent in civil life The causes of pneumothorax are —

1 EXTERNAL ORIGIN
   a PERFORATING WOUNDS
   b EXPLORING NEEDLE May prick lung, or diseased lung may rupture from rapid expansion after aspiration
2 LUNG PERFORATES INTO PLEURAL CAVITY —
   a TUBERCULOSIS OF LUNG – Commonest cause Usually rupture of cavity or a caseous focus in acute phthisis (In chronic forms adhesions and thickening usually protect)
   b NEOPLASMS Rarely GANGRENE, ABSCESS, BRONCHIECTASIS
   c SUDDEN STRAIN in normal persons No ill effects Rapid recovery Very rare
3 PLEURAL CONTENTS RUPTURE INTO LUNG Empyema
4 INFECTIONS OF PLEURA WITH ANAEROBIC GAS-FORMING BACILLI.—Very rare in civil life
5 NEOPLASMS OF ALIMENTARY CANAL PERFORATING INTO PLEURA, ABSCESS OF LIVER PERFORATING LUNG AND PLEURA SIMULTANEOUSLY —Very rare.

Affections of the Mediastinum—New Growths, *continued*

nerve, and heart (2) Involvement of lung, (3) Pleural effusion

2 COUGH — Often severe, with paroxysms May be brassy

VARIOUS OCCASIONAL PRESSURE SYMPTOMS —*Pain* over chest, may radiate to arms rarely as severe as aneurysm *Cyanosis Dysphagia, Alterations of voice* weakness or hoarseness Inequality of pulses and of pupils rare

Physical Signs.—*Posture* commonly sits up, with head thrown back Inspection important, but all signs may be absent *Cervical glands* may be enlarged Clubbing of fingers rare

INSPECTION AND PALPATION

1 RESULTS OF PRESSURE ON VESSELS Usually unilateral and in upper portion of body (1) Cyanosis (2) Superficial veins distended, may be collateral circulation (3) Œdema

2 VISIBLE TUMOUR (usually absent) May be in neck or after erosion of chest wall rarely May *pulsate*, transmitted from aorta or rarely a vascular tumour, but *not expansile*

3 IMMOBILITY OR BULGING OF AFFECTED SIDE

4 DISPLACEMENT OF HEART

PERCUSSION AND AUSCULTATION —Tactile fremitus breath sounds, and vocal resonance diminish and dullness increases as tumour nears chest wall May be systolic murmur at base

PLEURAL EFFUSION — Often present

INVASION OF SPINAL CORD AND MYELITIS Rare

Site of Tumours.—

1. ANTERIOR MEDIASTINUM Origin from connective tissue or thymus Special signs sternum dull on percussion pushed forward, œdema and pressure on veins common, cervical glands enlarged. Dyspnœa marked
2. MIDDLE AND POSTERIOR MEDIASTINUM Less common Origin from lymph glands Symptoms in excess of signs dyspnœa extreme ringing cough dysphagia
3. LUNG AND PLEURA Rapid emaciation Pressure signs slight Effusion early Cervical glands may be enlarged

Diagnosis (*see* THORACIC ANEURYSM) Often difficult Wassermann reaction, X ray, and exploring needle assist Also removal of an enlarged gland Diagnosis from

1 ANEURYSM Similarity is due to pressure effects

Note —IN TUMOUR — Cyanosis, pressure on veins, and pleural effusion more common

IN ANEURYSM —(1) Wassermann reaction always positive (2) X ray (3) Diastolic shock and loud aortic second sound (4) Expansile pulsation (5) Tracheal tugging

2. LARGE PERICARDIAL EFFUSIONS —Shape of dullness and weak heart-sounds.
3. PLEURAL EFFUSION
4. TUMOURS OF LUNG

Treatment.—Palliative.

2. VARIOUS AFFECTIONS OF THE MEDIASTINUM.

Lymphadenitis.

CAUSES—Inflammation of the glands near the bronchi, especially on the right, may be due to :—

1 TUBERCULOSIS—Constant in pulmonary tuberculosis May spread from cervical glands Not uncommon with no other focus

Temporarily :—

2 ACUTE FEBRILE CONDITIONS IN CHILDREN

3 INFLAMMATORY CONDITIONS OF THE LUNGS

SYMPTOMS Often absent or doubtful Slight unilateral changes in physical signs, percussion, and auscultation Suggested by spasmodic cough in children Eustace Smith's '*venous hum*', audible at root of neck only when head thrown back attributed to pressure of glands on large veins

Suppurative Lymphadenitis.

Abscess of tracheal or bronchial glands may occur (1) In tuberculosis (2) after simple adenitis Occasionally they rupture in various directions Tuberculous glands may inspissate

Abscess of the Mediastinum.

VARIETIES AND CAUSES

1 ACUTE (*a*) Trauma usual cause e g, foreign bodies swallowed bougies (*b*) Acute fevers

2 CHRONIC Tuberculosis

SYMPTOMS Pain behind sternum, with sweats and signs of sepsis

PHYSICAL SIGNS Rarely definite May be superficial œdema and dullness Rarely a tumour at sternal notch May rupture in any direction

In chronic cases, inspissation commonly results

Indurative Mediastino-Pericarditis.

A chronic fibrosis of the mediastinal tissues May be tuberculous or no cause apparent Rare Onset commences in youth, and the case may be slow progress (*See also* CHRONIC PERITONITIS) Three groups are described

1. ADHERENT PERICARDIUM WITH THICKENING OF MEDIASTINAL TISSUES—True indurative mediastino-pericarditis

   SYMPTOMS as in adherent pericardium with cardiac hypertrophy : dyspnœa, cyanosis, cardiac failure. Mediastinal friction, crackling along sternum on raising arms above head is ascribed to stretching adhesions There may also be chronic peritonitis, and the condition be part of a chronic *polyserrhomenitis* (*polyserositis*)

2. PERICARDITIS EXTERNA ET INTERNA—Pericardium adherent to sternum but mediastinum free

3. MEDIASTINITIS WITHOUT INVOLVEMENT OF PERICARDIUM.

Section VII—DISEASES OF THE KIDNEY AND URINARY TRACT.

CHAPTER LXXXV

MOVABLE KIDNEY.

(*Nephroptosis*)

Normal Position of Kidneys.—The kidneys are held in position by (1) The fatty capsule or 'perirenal fat' (2) Peritoneal pressure of viscera under tension of abdominal muscles, (3) Renal blood vessels Other factors are (4) Kidney rests in a fossa which is deeper in males than females and deeper on left than on right (5) Layer of areolar capsule is carried up to diaphragm and stronger on left, (6) Peritoneal reflections on front of kidney
Movable kidney results from changes in these factors wasting relaxation of abdominal muscles, possibly also congenital elongation of renal vessels, drag of loaded cæcum, and shallow fossa
As the kidney descends, the upper pole and outer edge tend to rotate forwards

Etiology.—

SEX —Nearly ten times commoner in women Common both in nulliparæ and multiparæ
AGE —Commonest 30 to 40 years
RIGHT KIDNEY much more frequently affected than left ascribed mainly to descent of liver on respiration
FREQUENCY IN WOMEN ascribed to
(1) Relaxation of abdominal muscles by pregnancy in neurotic women, and other causes
(2) Tight lacing compressing the lower thorax and also pull of heavy garments
(3) Occurrence of Glénard's enteroptosis neurasthenia with gastro intestinal disturbances specially common in thin, long chested women
DEGREES OF MOBILITY 'Movable kidney' is used for all degrees but the following are also described
1 PALPABLE KIDNEY —Lower pole palpable
2 MOVABLE KIDNEY On inspiration, fingers slip over upper pole
3 FLOATING KIDNEY —Freely movable about abdomen
The occurrence of a kidney with a true mesonephron is apparently apocryphal
In practice, amount of displacement and severity of symptoms are often unrelated

Symptoms.—Affect renal, gastro intestinal, and nervous systems When symptoms occur, trauma or strain may excite onset

TYPES.—

1. No Symptoms. In majority (80 per cent). When condition detected accidentally, do not inform patient
2. Lumbar Pain and dragging discomfort, or rarely intercostal neuralgia. Kidney often tender.
3. Gastro-intestinal Symptoms. *Often with neurasthenia* or hysteria Dilatation of stomach and gastric symptoms from drag on duodenum; constipation from interference with colon; jaundice and possibly gall-stones from pull on bile-ducts may occur *
4. Dietl's Crises. Severe attacks, simulating or identical with renal colic. Pain radiating down ureter, shivering, vomiting, scanty urine, perhaps hæmaturia. Local symptoms may be:
   a. Kidney tender, but no tumour, though attack may end with passage of much clear urine Possibly torsion of renal vessels
   b. Intermittent hydronephrosis. A renal tumour appears rapidly, disappears after a few days, with discharge of urine Ascribed to kinking of ureter Pyelitis or pyonephrosis may also occur

Visceroptosis, gastroptosis, etc, are frequently associated

Diagnosis.—Rarely doubtful.

FROM ENLARGED GALL-BLADDER. Distinct interval to palpation and percussion between liver and kidney, kidney can be pushed down

FROM OVARIAN TUMOUR Occasionally

Treatment.—

INDICATIONS According to types of symptoms

1. No Symptoms —No treatment
2. Renal Discomfort —Medical at least 3 months Surgical, often good results
3. With Neurasthenia, etc Solely medical Surgical, very bad results
4. Dietl's Crises. (*a*) With intermittent hydronephrosis: Surgical. Treat acute attack as renal colic. (*b*) Without intermittent hydronephrosis: Medical, at least 3 months

MEDICAL TREATMENT.—*Indications*, (1) Increase body-fat by full diet and tonics (2) Strengthen abdominal muscles by massage and by exercises (bending, etc, night and morning) (3) Neurasthenic type: Rest cure

Abdominal Belt Often gives great relief (by supporting muscles). Should be put on while recumbent or in knee-elbow position, and removed at night. *Pad* to hold up kidney: best is indiarubber bag with air or glycerin, fitted into abdominal belt. Doubtful if any pad can retain kidney in position.

* Every conceivable ill, including insanity, has been ascribed to movable kidney.

Movable Kidney—Treatment, *continued.*

SURGICAL TREATMENT.—Fixation of kidney (nephrorrhaphy)

INDICATIONS FOR OPERATION.—Genuinely severe symptoms due to mobility of kidney and in absence of definite enteroptosis and neurosis. Difficulty is to exclude presence of neurosis—i.e., to distinguish type 2 and type 3.

CHAPTER LXXXVI.

ANOMALIES OF THE URINARY SECRETION.

I. ANURIA.

In anuria, no urine enters the bladder. In retention the difficulty is to empty the bladder.

Causes.—These are: (1) Obstructive; (2) Non-obstructive.

1. OBSTRUCTIVE.—
   a. CALCULUS.—Common form. Calculus blocks one ureter, while other kidney is diseased. More rarely, calculi block both ureters.
   b. NEOPLASM, e.g., of bladder, compresses or involves ureters
2. NON-OBSTRUCTIVE. The causes are miscellaneous:—
   (a) Acute nephritis—in early acute congestion.
   (b) Acute fevers (usually temporary; rarely fatal).
   (c) Following operations on or injuries to the urinary system (from passage of catheter to nephrectomy).
   (d) Collapse stage of cholera and yellow fever (high mortality).

   More rarely:
   (e) Hysteria.
   (f) Poisoning with lead, phosphorus, or turpentine.

Symptoms in Prolonged Anuria.—

1. OBSTRUCTIVE FORMS.—('Latent uræmia'.) Usually no symptoms for several days. May be none until death. Usually slight drowsiness; pupils contracted; low temperature; slight twitchings; occasionally vomiting. Consciousness often until end. Death from cardiac or respiratory failure. Towards end may be ordinary uræmic symptoms. Duration 7 to 12 days or longer.
2. NON-OBSTRUCTIVE FORMS.—Symptoms of ordinary uræmia.

Treatment.—

1. OBSTRUCTIVE FORMS.—
   CALCULUS.—Operate on affected side. (Diagnosis as in nephrolithiasis.)
   NEOPLASM.—Usually non-operable.
2. NON-OBSTRUCTIVE FORMS.—Varies with cause.
   INDICATIONS are:—
   (a) To re-establish flow: Diuretics. Counter-irritants to

kidney mustard leaves, turpentine stupes, dry cupping. Open bowels with salines and enemata.

b. To maintain strength: Give stimulants, alcohol and strychnine.

c. To remove toxins: Stimulate skin with hot baths or packs, watching pulse for collapse (pilocarpine not advisable). Rectal saline injections, two pints four-hourly; or better, intravenous injection of normal saline, two pints, or of glucose (5 per cent solution); may be repeated after 4 hours.

DIURETICS:—

℞ Pot. Citratis	gr. x	Inf. Buchu	ad ℥j
Spt. Ætheris Nitrosi	ʒss		
	Four-hourly.		

Digitalis, diuretin, theocine sodium acetate, when flow recommences.

II. HÆMATURIA.

The presence of red blood corpuscles in the urine.

Etiology.—

1. RENAL CAUSES -
   - *a.* Nephritis, acute Less commonly granular kidney.
   - *b* Calculus.
   - *c.* New growths: often profuse
   - Rarer are:—
   - *d.* Renal infarct (from endocarditis).
   - *e* Early renal tuberculosis
   - *f* Certain poisons e g, carbolic acid, cantharides, turpentine
   - *g.* Angioma and capillary nævi of renal pelvis: usually profuse. Oxaluria may cause slight hæmaturia.
2. AFFECTIONS OF URINARY PASSAGES -
   - *a* URETER.—Calculus.
   - *b.* BLADDER.—(i) Neoplasms, papilloma or villous: ten profuse. (ii) Calculi: slight (iii) Bilharziasis: ery common in certain countries. (iv) Tuberculosis—rarely.
   - *c.* PROSTATE.—Tumour.
   - *d* URETHRA.—Calculus or gonorrhœa rarely
3. TRAUMA.—(Diagnosis of site of lesion important.)
4. GENERAL DISEASES.- Malignant specific fevers, and malaria rarely in blood and general diseases: purpura, leukæmia, scurvy.
5. ESSENTIAL HÆMATURIA ('Gull's renal epistaxis')—No recognizable cause. Rarely dangerous. Diagnose with caution.

Diagnosis.—Presence of blood in urine recognized by: (1) *Colour*: red or 'smoky'. (2) *Microscopy of deposit*: red cells present: trace of blood thus detected. Chemical test with guaiacum and ozonic ether less reliable. • Spectroscope will identify hæmoglobin, but not presence of cells.

• 'Smoky' tint due to acid salts of urine converting some blood pigment into acid hæmatin and methæmoglobin.

Hæmaturia, *continued.*

Differential Diagnosis of Cause.—

1. EXAMINATION OF URINE—
   (a) APPEARANCE OF URINE—
      Blood profuse usually calculus or neoplasm
      Colour bright red bladder or lower urinary tract
   (b) MICROSCOPICAL EXAMINATION Examine urine for casts and pus cells, when necessary also for tubercle bacilli *Bilharzia* ova
   (c) DISTRIBUTION OF BLOOD DURING MICTURITION Patient passes water into three vessels:
      Blood equally in all renal or severe vesical cause
      Blood mainly in first prostatic or urethral cause
      Blood mainly in last vesical origin
2. PHYSICAL SIGNS—Rectal examination for prostate—X rays Cystoscopy Catheterization of ureters
3. SYMPTOMS.—
   AGE—In elderly persons especially calculus or neoplasm In young persons may be tuberculosis
   DISTRIBUTION OF PAIN At end of penis cause in bladder Renal colic cause in kidney or ureter (Clots of blood from any site may cause pain in penis when passed)
   Various symptoms often point to cause of the hæmorrhage, e.g. endocarditis in renal infarcts

III. HÆMOGLOBINURIA.

A condition characterized by the presence in urine of free blood pigment without the corresponding presence of red cells

Methæmoglobin is almost always if not invariably present Hæmoglobin is present also relative proportions unknown

Etiology.—

(1) EXPOSURE TO COLD AND OVER EXERTION In individuals with poor circulation and cold extremities Allied to trophoneuroses and paroxysmal form
(2) SYMPTOMATIC Blackwater fever Yellow fever Rarely malaria, syphilis, severe burns, and infectious fevers
(3) TOXIC—Certain substances causing hæmolysis potassium chlorate, carbolic acid Other rare substances e.g. naphthol, arseniuretted hydrogen
(4) PAROXYSMAL HÆMOGLOBINURIA
(5) WINCKEL'S EPIDEMIC HÆMOGLOBINURIA (probably infective). Rare

Hæmoglobin from hæmolysis is converted normally into bile in the liver. When amount is excessive, some escapes into urine. Relationship between icterus and hæmoglobinuria is complex.

Character of Urine.—

COLOUR.—Almost black. Smoky if diluted.

ALBUMIN —Present, and may persist several days after disappearance of blood pigment

SEDIMENT —Profuse and dark Characterized by absence of sufficient red cells to account for pigment Contains débris of scanty red cells, urates

SPECTROSCOPE Methæmoglobin band between C and D, removed by Am S, also hæmoglobin bands (Urine usually needs considerable dilution)

Diagnosis.—Characteristic is presence of pigment in absence o sufficient red cells to account for the amount.

PAROXYSMAL HÆMOGLOBINURIA.

Connected with trophoneuroses and Raynaud s disease (*see* TROPHONEUROSES)

ETIOLOGY - Mainly in adult males Follows exposure to cold, especially with exertion, rare in warm climates

SYMPTOMS —Slight malaise, chill May be vomiting, diarrhœa, lumbar pain Pyrexia rare

DURATION Short one day Very rarely fatal

PATHOGENESIS *Hæmolysin* is present in the blood, and unites with red cells only at low temperature, but can attack cells of its own blood

TREATMENT Essential *warmth* and *hot drinks* Hæmolysis needs no further treatment Turpentine oil inadvisable

IV. ALBUMINURIA.

Albumin may occur in urine in the absence and without subsequent development, of the condition of nephritis

Varieties and Causes.—(1) Coarse renal lesions absent (2) Coarse renal lesions present Albuminuria of pregnancy may belong to either group

COARSE RENAL LESIONS ABSENT —

a. PHYSIOLOGICAL ALBUMINURIA —MacLean in apparently healthy recruits found albuminuria in 5 02 per cent viz, trace in 3 per cent, marked in 2 per cent, also casts in 2 per cent—viz, hyaline in 1 per cent, hyaline and epithelial in 1 per cent Of this total 1 per cent represented definite disease with inefficiency of kidney, remainder being harmless physiological albuminuria Age at discovery usually 15 to 30 years Generally discovered accidentally in routine school or life insurance examinations Hence more frequently recorded in males. May cease at puberty or persist longer Excretion may be erratic or permanent, or may follow severe exertion, exposure to cold, excessive protein diet, or conditions indicated by numerous names—e g, 'cyclic', present for a few days, then absent; 'orthostatic', on rising from bed—subjects usually neurotic. Also known as functional, postural, intermittent, dietetic paroxysmal, and as albuminuria of adolescence or of puberty.

Albuminuria—Varieties and Causes, *continued.*

B *Diagnosis only justified* when repeated examinations show (1) No other urinary abnormality, (2) Subject in good health, without renal, arterial, cardiac, or other disease. Evidence in such cases shows no shortening of life or subsequent nephritis, and the albuminuria is negligible (*See* TESTS OF RENAL EFFICIENCY, p 567)

b FEBRILE ALBUMINURIA —Transient trace common in severe pyrexias and at onset of specific fevers, especially pneumonia, diphtheria, scarlet fever, and typhoid No subsequent renal changes, differs from true nephritis occurring in later stages of fevers —e g, in scarlet fever

c PREGNANCY —Cause may be (1) Pressure of uterus, (2) Toxins acting on kidney

d PYURIA, HÆMATURIA

Of less practical importance are —

e BLOOD DISEASES —Trace often present in severe anæmia, leukæmia, etc

f POISONS – Arsenic, phosphorus, lead, mercury, turpentine, cantharides

g NUMEROUS DISEASES – Diabetes, gout, syphilis, exophthalmic goitre, Raynaud's and ancillary diseases, affections of nervous system, such as epilepsy, meningitis cerebral hæmorrhage

2 COARSE RENAL LESIONS AND CASTS PRESENT – –

a NEPHRITIS

b PASSIVE RENAL CONGESTION —From cardiac failure in diseases of heart and lungs Rarely from pressure on renal veins from neoplasm or thrombosis of vena cava

Prognosis.—Depends mainly on the progressive or non progressive nature, and on other signs of disease— renal arterial cardiac, etc

Trace of albumin after middle age needs careful life prognosis not unfavourable with soft arteries and no casts

For life insurance, reject or 'load' all cases except fully-proved physiological albuminuria

V. ALBUMOSURIA.

Of little importance Occurs with excessive cell destruction viz, in suppuration, pyrexia, resolving pneumonia, acute yellow atrophy, involution of uterus, and rarely in nephritis, especially syphilitic. Amount rarely large Presence often masked by concomitant albumin

TEST.—Not precipitated by heat after addition of acetic acid Cold nitric acid (or better, salicyl-sulphonic acid) causes a precipitate which dissolves on warming and reappears on cooling.

Multiple Myeloma. Kahler's Disease. (Bence-Jones' albumosuria.).—Two characteristics —

1. MULTIPLE TUMOURS OF BONE —Arise from bone-marrow, especially vertebræ, ribs, sternum; form large masses.

2. 'BENCE-JONES' ALBUMOSE' IN URINE.—Is correctly a protein; not normally produced in body. Excreted in large amounts (70 grm. daily). Precipitates at 50° to 56° C.; redissolves on warming; reappears on cooling.

Anæmia and cachexia occur. Death in about 2 years

VI. PYURIA.

Presence of pus in the urine.

Principal Causes.—

1. URETHRA.—Usually gonococcal. Rarely infections with *B. coli* and other bacteria.
2. BLADDER.—(1) Infections with *B. coli*; (2) Tuberculosis; (3) Calculi; (4) Neoplasms; (5) Prostatitis.
3. URETER.—Calculus.
4. KIDNEY.—(*a*) Pyelitis, pyelonephritis, pyonephrosis; (*b*) Calculus; (*c*) Tuberculosis; (*d*) *B. coli* infections.
5. RUPTURE OF EXTRANEOUS ABSCESSES.—Prostate, appendix, perinephric, etc. Usually large amount of pus for short time.
6. LEUCORRHŒA.—Few leucocytes.

Test.—Microscopic examination of deposit. (Phosphates, in alkaline urine, dissolve on addition of acid.)

For Bacteriology and Differential Diagnosis, *see* PYELITIS.

VII. LIPURIA.

The passage of urine containing *drops* of fat. Very rare.

Occurrence.—

1. EXCESSIVE INTAKE OR PRODUCTION OF FAT.—
   a. EXCESS IN FOOD, e.g., with cod-liver oil—'alimentary lipuria'
   b. LIPÆMIA of diabetes mellitus or, very rarely, acute leukæmia.
   c. PHOSPHORUS POISONING.
   d. PREGNANCY.
   Very rare: Fractures of long bones.
2. CHRONIC NEPHRITIS.—Rarely.
3. CHYLURIA.

Urine.—Turbid; drops of fat on surface, *clears with ether*, and fat can be recovered on evaporation of ether. (Beware of oil from catheter or addition of milk by patient.)

VIII. CHYLURIA.

Occurrence.—(1) *Filaria sanguinis hominis.* (2) Non-parasitic; extremely rare; may be obstruction to thoracic duct.

Urine.—Milky appearance. May be blood also. Sometimes clots to a jelly. Rarely drops of fat present.

IX. OXALURIA.

The presence in the urine of an abnormal number of calcium oxalate crystals. This is not necessarily a proof of excessive excretion.

Oxaluria, *continued.*

Principles of Excretion of Oxalates and of Oxaluria.—

1. Normal excretion is not more than 10 mgrm. *oxalic acid* daily.
2. Deposits form after certain vegetables, especially rhubarb, also spinach and tomatoes; but persistent deposits are pathological, in health there being a trace only.
3. Deposit is never heavy; crystals form on sides of glass; either octahedral or, less commonly, dumb-bell; always calcium oxalate. Urine containing crystals is acid, rarely neutral. (Normally, held in solution by acid sodium phosphate.)
4. Oxalic acid excreted is: (*a*) Mainly exogenous, taken in with the food, either (*i*) as oxalates, or (*ii*) from gastro-intestinal fermentation of purins; (*b*) Partly endogenous, since a trace persists in starvation. Oxalic acid given by mouth is excreted quantitatively.
5. Excretion said to be excessive with increased intestinal fermentation or absence of free HCl from gastric juice
6. Administration of free HCl increases absorption of calcium oxalate from food and excretion in urine.

Pathological Conditions connected with Oxaluria.—

1. CALCULI.—Oxalates (always calcium salt) are commonest constituents of renal and ureteric calculi.
2. HÆMATURIA AND PYURIA.—Every other cause must be excluded before ascribing these to oxaluria.
3. OXALIC ACID DIATHESIS.—Nervous dyspepsia, general irritability, depression, and neurasthenia, are associated with oxaluria Symptoms probably depend on metabolic disturbance also causing oxaluria.

Treatment.—Regulate bowels and digestion. Give magnesium, which hinders precipitation—viz, salts, farinaceous foods, beans, and peas. Avoid calcium foods—e.g., milk, eggs, and oxalate-rich vegetables.

X. CYSTINURIA.

Very rare. Many subjects are children of first-cousin marriages, may be hereditary, but rare. Commoner in males. Continues throughout life, but possibly intermittently.

Symptoms.—No general symptoms, but insolubility leads to formation of calculi.

Calculi.—Large, smooth, typical *soapy* feel. Cystin contains sulphur. *burns* with a blue flame without melting.

Urine.—Colour normal. Contains cystin crystals. Cadaverin and putrescin frequently also present; rarely leucin and tyrosin.

CHARACTERISTICS OF CRYSTALS.—Regular hexagonal plates; soluble in ammonia or HCl, insoluble in water, ether, and acetic acid.

Pathogenesis.—A 'chemical malformation' (Garrod) of amino acid metabolism A cystinuric usually metabolizes ingested cystin to sulphate, as do normal men, hence excreted cystin arises from protein of tissues and not of food; exceptions to this occur.

XI. PHOSPHATURIA.

Generally applied to deposit of phosphates in urine Excretion not necessarily increased, nor does an increased secretion necessarily, or even usually, lead to a deposition

General Principles of Excretion and Precipitation of Phosphates.—

1. Phosphates are excreted as (a) Alkaline phosphates, sodium and potassium, never precipitated as such, form three-fourths. (b) Earthy phosphates, calcium and magnesium, only soluble in acid urine, form one fourth total 2 to 5 grm daily
2. Origin (a) Exogenous, from food, (b) Endogenous, from nuclear tissue (nuclein, lecithin)
3. Precipitate of phosphate soluble on adding acid forms on heating urine even if acid, this is due to decomposition of two molecules of calcium hydrogen phosphate into one of mono calcium hydrogen phosphate and one of tricalcium phosphate ($Ca_3(PO_4)_2$), latter being comparatively insoluble in water
4. Physiological phosphaturia occurs after meals ('alkaline tide'), due to gastric secretion of HCl especially after rich protein meals, or with quantity of vegetables Results from increased excretion of 'fixed alkali' (sodium, potassium salts)
5. Phosphates deposit in presence of 'volatile alkali', viz, ammonia
6. Nature of precipitate
   - *a* 'Triple phosphate' ammonium magnesium phosphate
     Shape coffin lid', in very alkaline urine, feather- or fern shaped.
   - *b* 'Stellar phosphate' calcium hydrogen phosphate
     Shape long flat prisms, often in bunches, may occur in slightly acid urine Rare in health, occurs in diabetes and cachexia
   - Amorphous phosphates calcium and magnesium phosphates.

Pathology of Phosphate Sediments.—

1. IN ALKALINE URINES Importance depends on primary cause, e g, cystitis, in this, ammonia is due to bacteria decomposing urea
2. IN NERVOUS DISORDERS, especially of sexual organs — Often passed at end of micturition and mistaken for spermatozoa Little-known condition
3. IN CHILDREN —Calcium in urine increased probably an error of intestinal mucous membrane preventing normal excretion by the colon
4. PHOSPHATIC DIABETES —Excretion greatly increased, polyuria and emaciation; may closely simulate diabetes (but no glycosuria). Rare. Pathology unknown. Many die of phthisis

Anomalies of the Urinary Secretion, *continued.*

XII. INDICANURIA.

Indican is a compound formed in the tissues of potassium sulphate with indoxyl, derived from indole, a product of bacterial fermentation of proteins With strong acids, indican oxidizes to indigo, thus forming purple ring in urine floating on nitric acid depth of ring is approximate guide to quantity usually a trace

EXCESS suggests increased intestinal fermentation Occurs in constipation, mental depression, and occasionally in empyemata and unopened abscesses

XIII. BLACK URINES. MELANURIA.

Urine may be clear on passage, and become dark on standing All forms of *black* urine are rare Dilution of urine often gives guide to cause Very dark urine may occur in -

1. JAUNDICE —Only if very excessive
2. HÆMOGLOBINURIA May be extremely dark, on dilution tint is red
3. HÆMATURIA Rarely very dark, on dilution tint is red
4. HÆMATOPORPHYRINURIA —May be extremely dark on dilution tint is red
5. DRUGS —Especially resorcin Also carbolic acid ('carbo luria' colour due to hydroquinone)
6. BERRIES - Dark cherries, etc Colour rarely very marked.

In the following, colour develops on standing -

7. MELANURIA -On standing, or addition of nitric acid to oxidize Confined to melanotic sarcoma Colour is *black* true melanuria
8. ALKAPTONURIA On standing or addition of alkali
9. INDICANURIA —*Very rarely* is sufficient to darken urine

XIV. ALKAPTONURIA.

Due to excretion in urine of 'alkapton,' homogentisic acid (hydro quinone acetic acid) A harmless congenital 'chemical malformation (Garrod), metabolism of tyrosin being arrested at a certain point

Characteristics.-

1. Often several members of family, not hereditary, consanguineous marriage frequent
2. Dates from birth, noticed by staining of linen, commoner in males
3. No symptoms or effect on health May produce *ochronosis* staining of cartilages
4. *Urine,* normal when passed, darkens on standing or addition of alkali

   Reduces Fehling, but optically inactive, and does not ferment

   Drop of ferric chloride produces transient deep-blue colour

5. Tyrosin increases output in normal persons has no effect

No treatment necessary or effectual

Note —The statement formerly made that another acid, uroleucic acid, was also present, was based on a misapprehension.

XV. PNEUMATURIA.

Passage of gas with urine. Occurs in :—

1. **Gas-forming Organisms in Bladder.**—Usually introduced by catheter. Most common is *yeast fungus*, with *glycosuria*.
2. **Vesico-enteric Fistula.**

XVI. HÆMATOPORPHYRINURIA.

Cause. Usually due to *sulphonal*, rarely trional: long administration, generally females. Is iron-free hæmatin.

Urine.—Almost black, or deep port-wine colour, on passage.

CHARACTERISTICS.—(1) Does not give blood tests. (2) No albumin present. (3) Spectroscope: spectrum resembles hæmoglobin. *Colour is not due to hæmatoporphyrin*, for this is colourless in urine, and after its removal urine remains dark, but to some unknown pigment which accompanies it.

Prognosis.—Serious if drug is continued.

XVII. BLUE AND GREEN URINES.

Blue urine is invariably due to methylene blue; also
Green urine, except for special dark green tint occasionally seen in jaundice.

XVIII. OTHER SUBSTANCES.

Glucose, Acetone (*see* DIABETES). **Bile** (*see* JAUNDICE). **Leucin** and **Tyrosin** (*see* ACUTE YELLOW ATROPHY).

CHAPTER LXXXVII.

URÆMIA.

A toxæmia which may occur in any form of nephritis or anuria. Thus it may complicate: acute, chronic, or consecutive nephritis, amyloid kidney, tuberculous, cystic, and calculous kidneys; anuria from calculi, or following operations on the urinary system; hydronephrosis. Most common in acute and chronic nephritis.

Theories of Origin.—

1. Accumulation in blood of substances normally excreted by kidneys. In uræmia, urea in blood may rise from 0·015 to 0·2 per cent. or higher.

   Objections—(a) Suppression results in latent, but not acute or chronic, uræmia; (b) Urea and other known substances in urine do not experimentally produce uræmia and are not invariably increased in uræmia.

2. Decomposition in body of such substances, forming toxic products.

Uræmia—Theories of Origin, *continued.*

(3) Abnormal products from disturbed renal metabolism. Bradford showed that destruction of renal tissue results in increased excretion of urine with rapid protein metabolism.

(4) Localized œdema of brain. Could not account for all symptoms.

Symptoms.—Three types of uræmia may be recognized clinically: (i) Acute—in any nephritis. (ii) Chronic—especially in granular kidney. (iii) Latent—in anuria. Latent uræmia differs from other forms, and it is described separately (*see* ANURIA, p. 556).

There are three principal groups of symptoms: (1) Cerebral; (2) Gastro-intestinal; (3) Dyspnœic. They frequently co-exist, and cerebral symptoms are almost invariably present before death.

1. CEREBRAL SYMPTOMS AND TYPES—

a. COMA.—Most common type of uræmia. Usually terminates the other forms. Gradual or rapid onset. Often preceded by *headache, drowsiness,* or *delirium,* by *vomiting, twitching of muscles,* especially extensors of wrist, and *cramps in calves.* Tongue furred and breath heavy. Pupils contracted. Knee-jerks increased. Temperature subnormal. May last several days, rarely longer.

b. EPILEPTIFORM CONVULSIONS. Onset (i) abrupt, or (ii) preceded by headache and restlessness. Fits usually general, resemble epilepsy (cry rare), and may recur rapidly. Unconsciousness invariable during general convulsion: also usually in intervals when recurrent, but may be incomplete. Temperature usually low, but may rise. Uræmic amaurosis may follow—viz., blindness without retinal changes.

Less common are:—

c. ACUTE MANIA.—May occur without warning. Usually end in coma.

d. DELUSIONAL INSANITY ('folie brightique'). Granular kidney, otherwise unsuspected, is often found in asylum post-mortems.

NERVOUS SYMPTOMS RELATED TO URÆMIA—(1) Headache—early and often severe. (2) Tingling and numbness of fingers. (3) Twitching of muscles. (4) Cramps in muscles, especially calves. (5) Itching of skin. Less commonly: (6) *Local and transient paralyses*: any form of hemiplegia or monoplegia may occur (post mortem—localized œdema of brain, but no gross lesion). (7) Transient amaurosis or deafness. (8) Persistent insomnia; may continue until death, with few other symptoms.

2. GASTRO-INTESTINAL SYMPTOMS.—Uncontrollable vomiting, nausea, and hiccough. More rarely diarrhœa. Later follow uræmic dyspnœa and cerebral symptoms, ending in coma.

3. URÆMIC DYSPNŒA.—Usually nocturnal. Breathing hissing and noisy. May be continuous, or—

CHEYNE-STOKES RESPIRATION.—Commoner in chronic uræmia; may persist for months. With coma is of grave prognosis.

SKIN.—A deposit of urea may form ('urea frost').

Summary of Symptoms frequently occurring in Acute Uræmia.—

EARLY STAGE Headache Sleeplessness Nausea and vomiting Tingling of fingers Slight twitching of muscles Cramps in calves Breath heavy Tongue furred Pupils contracted

PROGRESSING — Severe vomiting Paroxysmal dyspnœa Various paralyses

LATE STAGE.— Cheyne Stokes breathing Epileptiform convulsions Death in coma

Chronic Uræmia.—A term applied to the persistent presence of the milder symptoms —e g, headache occasional vomiting, slight twitching of muscles, restlessness insomnia, etc Wasting, dry skin, and sallow complexion usually present May continue for many weeks Concludes by —

1 Terminal infections, i e common acute pericarditis, pleurisy, rarely peritonitis, endocarditis, and meningitis
2 Acute uræmia
3 Temporary improvement

Diagnosis.— See COMA p 294

Prognosis.—Always serious Recovery more frequent in acute nephritis than in chronic forms From epileptiform convulsions in acute nephritis and pregnancy recovery is not uncommon, but when occurring in chronic nephritis recovery is very rare

Treatment.—See CHRONIC INTERSTITIAL NEPHRITIS p 579

CHAPTER LXXXVIII.

TESTS OF RENAL EFFICIENCY.*

Numerous tests of renal function are now being studied. This applies to nephritis eclampsia, and surgical diseases of the genito-urinary tract No single test is reliable except in extreme instances. Two or preferably three should be performed, and considered in conjunction with clinical manifestations Cardiac failure will influence any of the tests

Blood Urea.—In certain renal disturbances, nitrogenous bodies are retained in the blood These include urea, uric acid, creatinin, and purin bodies, but estimation of urea is sufficient guide Is most valuable single test, but retention does not occur until most of kidney fails to function

Note —Reduction in diet will of itself lower the blood urea, but such an effect is no evidence of improvement

NORMAL —Urea 15 to 40 mgrm per 100 c c. of blood (0·015 to 0 040 per cent). The higher figures apply to later life Over 50 mgrm is evidence of disturbed renal function.

ACUTE NEPHRITIS.—Estimations every ten days form guide to progress

* See especially MacLean, *Renal Disease* (Constable & Co.)

Tests of Renal Efficiency—Blood Urea, *continued.*

LARGE WHITE KIDNEY.—Blood urea normal.

CHRONIC INTERSTITIAL NEPHRITIS.—Amount tends to be increased. Is guide to prognosis.

SURGICAL DISEASES OF GENITO-URINARY TRACT.—Test is of great value, especially when considered with 'urea concentration factor'. If over 80 mgrm., operation is dangerous.

AMBARD'S COEFFICIENT OF UREA EXCRETION.—Practically depends on blood urea. Little used except in France.

2. **Urea Concentration Test'** (MacLean and de Wesselow).—Reveals slighter lesions than blood urea. Method: Administration of 15 grm. of urea in 100 c.c. of water, patient emptying his bladder just previously. At end of one hour, urine is passed and the urea estimated. If urea is 2 per cent or over, the kidney is acting efficiently. If below this percentage, the urine is examined again at the end of two hours; if still below 2 per cent, the kidney action is deficient, roughly proportional inversely to the percentage of urea.

   Amount of urine must not exceed 120 c.c. Diuresis, due to urea, occasionally occurs, and in such case results are not necessarily evidence of renal inefficiency.

'UREA CONCENTRATION FACTOR.'—Found by comparison of blood urea with urine urea, i.e., test of concentration by kidney. Normal concentration is 70 to 100 times. Valuable in surgical conditions.

3. **Phenolsulphonephthalein Tests.**—Subject drinks a glass of water and empties the bladder; 1 c.c. of standard solution of dye injected into lumbar muscles. Bladder emptied again one and two hours later. Amount of dye passed estimated quantitatively with Duboscq colorimeter. Normally 70 per cent in two hours. Under 50 per cent is evidence of renal inefficiency. None may appear.

   Difficulties arise from presence of blood, urinary pigments, and technique of estimation, but results satisfactory to many workers. With catheterization of ureters, long duration of test is also a disadvantage.

4. **Indigo-carmine Test.**—Only employed in surgical conditions, for testing relative working of each kidney. Inject intravenously 2 c.c. of 0·4 per cent solution. Blue urine should appear in 7 minutes.

5. **Diastatic Test.**—Diastase from pancreas enters blood and is secreted into urine. Measured by hydrolysing action on starch. Normal: 6 to 20 'units'. Often reduced, or absent, in renal inefficiency, but test apparently unreliable.

   Numerous other tests exist: Estimation of urea excretion by catheterization of ureters (comparison of kidneys). Leathes' water test (reliability doubtful). Chloride test (in parenchymatous nephritis; needs a constant diet and chloride equilibrium).

CHAPTER LXXXIX.

ACUTE NEPHRITIS.

(*Acute Bright's Disease.*)

Acute inflammation of the kidneys, changes occurring in the epithelial, vascular, and intertubular tissues, and associated with albuminuria and œdema.

Etiology.—

1. COLD.—Chill is an important exciting factor: probably some other cause, exciting or predisposing, must also be present.
2. SPECIFIC FEVERS.—Particularly scarlet fever, less commonly enteric, measles, diphtheria, and rarely others. Also septicæmia, secondary syphilis, acute tonsillitis, and rarely acute tuberculosis.
3. TOXIC AGENTS.—Drugs such as turpentine, potassium chlorate, cantharides, carbolic acid.
   Experimentally: certain bacterial toxins; also vinylamine. Alcohol, lead, and mercury cause chronic, but probably not acute, nephritis.
4. PREGNANCY.—Eclamptic and toxæmic kidneys have special characteristics, not here referred to.
5. EXTENSIVE SKIN LESIONS.—Burns, chronic diseases, erythemata, etc. Rare.
6. EPIDEMIC NEPHRITIS.—Prevalent in the war (*see* p. 572). Probably infective, transmitted by lice. But acute nephritis of civil life cannot be regarded as infectious.
   In majority of cases, no cause is recognizable.

Morbid Anatomy.—

MACROSCOPIC.—Kidneys large, deep red, capsule strips easily, leaving marbled surface and prominent stellate vessels.

ON SECTION.—Marked congestion. Cortex swollen. Glomeruli often prominent. Differentiation of cortex and medulla distinct. Pyramids deep red.

HISTOLOGY.—

1. GLOMERULI.—Proliferation and desquamation of lining epithelium. Capsule contains blood-cells. Vessels of tuft often thrombosed.
2. TUBULES.—Degeneration and desquamation of epithelium and necrosis: in places masses of various cells and casts. Blood-cells often present. Swelling of kidney mainly due to dilatation of tubules.
3. INTERSTITIAL TISSUES.—Inflammatory exudate: blood-cells, or small round cells. Vessels dilated

Acute Nephritis—Morbid Anatomy, *continued.*

In scarlatinal nephritis changes may affect mostly or even solely the glomeruli, a pure acute glomerulitis, but often the other structures are also affected.

Symptoms.—

MODE OF ONSET.—Variable. In children, and following chill, often rapid; in specific fevers insidious. Typical symptoms commoner in children; in adults often slight malaise with severe urinary changes.

SYMPTOMS AT ONSET.—(1) Headache; (2) Puffiness of eyes and face, and of ankles; (3) Nausea and vomiting; (4) Urine diminished and altered. General malaise. Constipation. Temperature 101°–103°; in adults may be apyrexial. Complexion pasty; skin dry; tongue furred; pulse not specially rapid. May be rigors or, in children, convulsions. Dyspnœa not common in adults.

CONDITION DEVELOPED.—

ŒDEMA.—Chiefly affects subcutaneous and loose areolar tissues: sacrum and scrotum common. May be universal. Pleural and peritoneal exudations and œdema of lungs occur, but not so frequently as in cardiac dropsy.

ANÆMIA.—Early and severe. Mainly from hydræmia.

OCULAR CHANGES.—Albuminuric retinitis: is rare in acute nephritis, except with pregnancy.

CARDIAC CHANGES.—Blood-pressure may be increased.

URINE.—

QUANTITY.—Scanty. Often a few ounces daily.

COLOUR.—Deep or smoky (blood).

SPECIFIC GRAVITY.—1025 to 1035.

ALBUMIN.—Large quantity.

UREA.—Percentage high. Daily excretion low.

DEPOSIT.—Blood-cells; hyaline, granular, epithelial, and blood casts; much débris.

CHLORIDES.—Trace only.

Progress.—In favourable cases, improvement after few days to one to two weeks; increase in amount of urine, excretion of urea and chlorides, diminution of œdema, and fall in blood urea (and often blood-pressure) usually run parallel; polyuria often marked—ascribed to diuretic action of blood urea.

Termination.—

1. RECOVERY.—Not infrequent in cold and syphilitic forms.
2. CHRONIC NEPHRITIS.—Frequent sequel in adults: trace of albumin permanent: attacks of subacute nephritis recur.
3. URÆMIA.
4. PERICARDITIS, PNEUMONIA, PLEURISY may occur and be fatal. Rarely, ACUTE CARDIAC DILATATION.

Prognosis.—Serious symptoms are: marked diminution of urine, low arterial tension, uræmia, serous effusions. Convulsions at

onset in children are not especially serious. Condition usually becomes chronic if albumin present after one month: almost invariably if present after three months. Severity of œdema and albuminuria at onset affects prognosis, but rec very may occur with any degree. Fall in blood urea and rise of urea in urine may indicate good final prognosis, even with clinical condition stationary. In syphilitic nephritis, especially, albuminuria is intense, with few other symptoms; reacts rapidly to specific treatment (*see* RENAL SYPHILIS, p. 272).

Diagnosis.—Usually simple. Difficulties arising are mainly due to not examining urine: symptoms may thus suggest anæmia, acute gastritis (vomiting), various cerebral conditions (headache and vomiting). Symptoms may be very slight in adults a puffiness of the eyes noticed by friends—even with marked albuminuria.

OTHER FORMS OF ALBUMINURIA causing difficulty:—

1. Febrile albuminuria is not necessarily acute nephritis.
2. Passive congestion of kidneys or renal infarct in heart disease.
3. Acute exacerbation in chronic nephritis. Cardiovascular and ocular changes present.

(*Very rarely*, typical symptoms occur without albuminuria.)

Treatment.—*Indications*: (1) Rest kidneys by (a) Dieting; (b) Utilization of skin and bowels for excretion. (2) Treat symptoms.

GENERAL HYGIENE.—Keep patient in bed until condition has disappeared, between blankets and clad in flannel.

DIET.—

AT ONSET. Strict milk diet. (Anorexia usual.) Milk only (1½ to 2 pints) for not more than ten days for adults. If desired, may be flavoured with coffee.

PROGRESS.—Thicken milk with arrowroot; then gruel and arrowroot, bread and butter and fruit. Avoid meat extracts. No alcohol. *Increase of diet* to be gradual, especially with meat, changes being guided by the urine as well as by symptoms. If with lapse of time more food is necessary though œdema persists, give salt-free diet, since chlorides in case œdema: bread, eggs, rabbit, and vegetables, avoiding milk.

ORDER OF ADDITIONS TO MILK.—Farinaceous food; bread and butter. Eggs. Fish and vegetables. Chicken. Meat.

FLUID.—Give alkaline drinks freely, such as lemonade and potus imperialis (acid potassium tartrate ʒ ij, lemon-juice ℥ss, syrup ℥ss, water to a pint). If necessitated by vomiting, give fluid by bowel.

EXCRETION BY SKIN.—Sweating encouraged by hydrotherapy, daily hot bath, wet pack, or hot-air bath (first bath not exceeding 120 to 140° F., never exceeding 170°); duration 15 to 20 minutes; watch pulse and stop on weakening: sweating is assisted by hot drinks; injection of pilocarpine (gr. ⅙ for adult, gr. $\frac{1}{20}$ to $\frac{1}{14}$ for child) may be given at commencement. If pilocarpine causes bronchorrhœa, do not repeat.

Promotion of sweating often difficult, especially in dry skin of uræmia.

Acute Nephritis - Treatment, *continued.*

EXCRETION BY BOWELS.—Must be freely open. Give morning purges: sulphate of magnesium (ʒj or more in little fluid); for children, fluid magnesia. Pulvis jalapæ co. (ʒss for adult man) or pulvis elaterinæ co. also good.

DRUGS.—No drug directly controls kidney changes or influences albuminuria. As diuretics, digitalis or strophanthus may be used when acute symptoms have cleared and blood-pressure not high. With other diuretics caution is necessary; diuretin, theocin sodium acetate, and caffeine citrate sometimes used for œdema.

SPECIAL SYMPTOMS—

VOMITING.—Often troublesome. Ice to suck. Tinct iodi (♏j in water ℥ss, hourly), or dilute hydrocyanic acid (♏iij, t d.s). Restrict food. Fluid by rectum.

ŒDEMA.—Specially encourage excretion by skin (hydrotherapy) and bowels. If excessive, multiple incisions of skin of legs, or insert Southey's tubes in dorsum of feet: asepsis important owing to sodden tissues. Pleural exudate and ascites (rarely) may need removal, if causing symptoms from pressure. Do not stint fluid, but avoid unnecessary amounts. Loss of weight is valuable but inconvenient measure of improvement. Increase in urine simpler.

URÆMIA.—*See* CHRONIC NEPHRITIS, p 573

ANÆMIA.—After acute symptoms have subsided, *give iron*: for adults, perchloride (tinct ferri perchlor. ♏x); for children, phosphate. Alginoid iron is a good preparation, not causing constipation.

PERSISTENCE OF ŒDEMA.—Treat by Epstein's diet (*see* CHRONIC PARENCHYMATOUS NEPHRITIS, p 575).

CONVALESCENCE.—Avoid chills. Give tonics. Keep bowels open. Moderate diet.

CLIMATE FOR WINTER.—In England: South-west coast. Abroad: West Indies, Madeira, and Canaries. America California.

WAR NEPHRITIS.*

Acute nephritis began to affect large numbers among the troops in France in March, 1915, and remained prevalent until the end of the war. It was almost, but not entirely, confined to troops in the front line.

A SPECIFIC INFECTIOUS CAUSE is accepted as probable, possibly transmitted by lice. Infection theory agrees with following phenomena: (1) A unit with a high incidence at the front continues to produce cases for some time when in rest: i.e. when once established the disease persists apart from locality. (2) The nephritis develops within a comparatively short period of the man's arriving at the front: more than half the cases occur within three

* MacLean, *Report on War Nephritis* (Medical Research Committee). This report contains much research and information on nephritis of great value.

months and nearly all within six months (3) Certain localities were more affected than others, and units showed more cases after entering these areas

The general condition resembles civilian nephritis except for the constancy of marked dyspnœa

Progress is usually rapid and favourable Œdema and albuminuria generally diminish rapidly accompanied by well marked diuresis fall in blood pressure and body weight, and rise in percentage of hæmoglobin in the blood Disease tends to be considerably milder than civilian nephritis

CHAPTER XC.

CHRONIC NEPHRITIS.

(*Chronic Bright's Disease*)

CLASSIFICATION.

Three forms may be distinguished —

(I) **Chronic Parenchymatous Nephritis: Large White Kidney.** Characterized (*a*) Clinically by œdema—i e "wet" or hydræmic type of nephritis (*b*) Chemically by retention of chlorides, by normal blood urea and normal renal efficiency tests

(II) **Chronic Interstitial Nephritis: Red Granular or Arteriosclerotic Kidney.**—Characterized (*a*) Clinically by cardiovascular changes and uræmic manifestations (*b*) Chemically by retention of nitrogenous products by increased blood urea, and positive renal efficiency tests—i e, the "dry" or azotæmic type of nephritis

Above two forms often typical clinically, chemically and pathologically summary (adapted from MacLean) —

		Large White Kidney	*Granular Kidney*
1	Œdema	Present	Absent
2	Cardiovascular changes	Absent or slight	Marked
3	Albumin	Marked	Slight may be absent
4	Chlorides in urine	Trace or absent	Normal
5	Urea concentration	Normal .	Diminished
6	Blood urea .	Normal . .	Increased
7	Diastatic reaction	Nearly normal .	Low
8.	Uræmic symptoms	Unusual .	Common

(III) **Small White Kidney.**—This form is varied in its manifestations —

Clinical course may be —

(1) Identical with large white kidney.

Chronic Nephritis, *continued.*

(2) May commence as in large white kidney, and develop, sometimes but not always after repeated attacks, the clinical characteristics of red granular kidney.

Note.—This sequence may occur with typical small white kidney. But, in addition, all intermediate forms occur pathologically between large and small forms: relationship has been much discussed. Sufficient time has not elapsed to correlate renal efficiency tests with post-mortem findings.

(3) Identical from onset with red granular kidney: Bradford's 'primary small white kidney'. Duration short and pathological changes marked. Cardiovascular changes extreme (varying, even marked, degrees occur in previous groups). Is an entity apart from red granular kidney, differing in: (i) Morbid anatomy. (ii) Age: occurs in young adults, very rare over 30 years. red granular kidney, very rare under 40 years.

Amyloid kidney is not a form of chronic Bright's disease.

I. CHRONIC PARENCHYMATOUS NEPHRITIS.

(*Large White Kidney.*)

Etiology.—

1. Sequel of acute nephritis of cold, fevers (especially scarlet fever), and pregnancy.
2. Insidious onset.
3. Lead and possibly alcohol.

AGE.—Especially children and young adults.

Morbid Anatomy.—

KIDNEY.—

MACROSCOPIC.—Large kidney (6 to 9 oz each). Capsule thin, strips readily. Surface smooth and pale. Stellate veins injected.

On Section.—Cortex swollen and opaque yellowish-white Demarcation distinct. Pyramids usually congested

HISTOLOGY.—

a. Glomeruli.—Enlarged. Glomerulitis. Capsules thickened. Epithelium degenerated. Hyaline degeneration of capillaries.

b. Tubules.—Changes prominent. Epithelium desquamated; hyaline and granular degeneration. Tubules in places distended with masses of such cells; in other places empty.

c. Interstitial Tissue and Arteries.—Changes slight.

Description applies to typical specimens. The size of the large white kidney is mainly due to the accumulation of cells in the tubules. The smallness of the small white kidney is partly due to the shedding of tubular epithelium. Inter-

mediate forms common. Thus some authorities regard the small white as a later stage of the large white kidney.

OTHER ORGANS.—Cardiac hypertrophy and thickening of arteries slight.

Symptoms.—

MODE OF ONSET.—

1. INSIDIOUS.—Common. Initial symptoms: puffy eyes or feet, dyspepsia, pallor, wasting.
2. SEQUEL TO ACUTE NEPHRITIS.

CHIEF SYMPTOMS AND SIGNS.—General resemblance to acute nephritis.

1. ŒDEMA.—Early, marked, and obstinate. Earliest in face (in morning) and feet. May be general, involving serous membranes. Ascites very common.
2. GASTRO-INTESTINAL SYMPTOMS. Nausea; anorexia; vomiting (may be serious); more rarely diarrhœa.
3. ANÆMIA, WEAKNESS, and WASTING.—Last may be masked by œdema.
4. HEADACHE.
5. TONGUE furred, BREATH foul. PYREXIA: rarely exceeds 101° in absence of complications.
6. URÆMIC SYMPTOMS AND URÆMIA.—Not common. Cardiovascular changes unusual or slight.

URINE—

SPECIFIC GRAVITY.—1020 to 1035.

COLOUR.—Turbid with urates, or smoky with blood.

QUANTITY.—Reduced (20 to 25 oz.). Varies with œdema, increase pointing to improvement. Diminishes with increase of œdema or onset of uræmia. Varies also with vomiting, purging, and diet (increased by milk).

ALBUMIN.—Abundant. Frequently 1 per cent. Rarely 5 per cent.

DEPOSIT.—Casts of various sizes and kinds—hyaline, granular, epithelial, and fatty; leucocytes, often numerous, may be red cells.

CHLORIDES.—Greatly diminished. May be trace only.

RENAL EFFICIENCY TESTS AND BLOOD UREA.—Normal.

Prognosis.—Always grave. Unfavourable features are: persistent œdema, all complications. Recovery may occur after long duration.

Mode of Death and Complications.—

1. General anasarca and œdema of lungs. Cardiac failure.
2. Pneumonia, pleurisy, and pericarditis.
3. Uræmia.

Treatment.—In general resembles acute nephritis.

EPSTEIN'S DIET.—If œdema persists, treatment on Epstein's lines often effective. Basis: (1) High protein diet; (2) Minimum of salt; (3) Little fat, moderate carbohydrates.

DIET.—Protein 120 to 240 grm.; fat 20 to 40 grm.; carbohydrates 150 to 300 grm. Articles: lean veal and ham,

Chronic Parenchymatous Nephritis—Treatment, *continued.*

whites of eggs, oysters, jelly, Lima beans, lentils, peas, rice, oatmeal, mushrooms, bananas, skimmed milk, coffee, tea, and cocoa. Fluid: 1200 to 1500 c.c.

Œdema often subsides rapidly, with general improvement: amount of albumin passed usually unaffected.

EPSTEIN'S THEORY.—(1) Excretion of protein in urine reduces protein content in blood; by giving a high protein diet, content is increased and resulting greater osmotic pressure draws fluid from tissues and enables secretion by kidneys.

(2) Blood in nephritis shows increase of lipoids.

Theory not generally accepted, and good results ascribed to diuretic action of increased urea in blood.

ADMINISTRATION OF UREA.—Urea by mouth (30 grm. daily) often produces great increase in urine and diminution in œdema (MacLean and Russell).

II. CHRONIC INTERSTITIAL NEPHRITIS.

(*Red Granular Kidney. Arteriosclerotic Kidney. Gouty Kidney.*)

An increase of fibrous tissue in kidneys with ancillary parenchymatous changes. Cardiovascular changes marked. Uræmic symptoms common.

Two Forms of chronic interstitial nephritis may thus be recognized:—

1. PRIMARILY RENAL.—The kidneys are small, changes extreme; the arteriosclerosis is not extreme, though definite. This is the typical 'red granular kidney'.
2. ADVANCED ARTERIOSCLEROTIC CHANGES.—The kidneys are about normal size, though with distinct fibrotic changes. This is the 'arteriosclerotic' kidney, the circulatory changes being primary.

The clinical differences between these forms are slight.

Sclerosis of the kidneys also occurs (1) as a senile change, (2) in 'small white kidney'. The latter form is described below, and is not referred to here.

Etiology.—

AGE.—Rare before 40 years. Commoner in males.

NO PREVIOUS RENAL DISEASE.

CONTRIBUTORY CAUSES.—Excesses: over-work, over-eating, alcohol; gout, syphilis, and lead.

HEREDITY.—Is certainly a factor, families being liable to early arterial degeneration.

IN CHILDREN.—Instances very rare, but usually adva[illegible], probably usually inherited or syphilitic; in extreme form in progeria. Also in renal infantilism.

Morbid Anatomy.—

KIDNEYS:—

MACROSCOPIC.—Small (1½ to 3 oz. each). Red colour. Capsule adherent. Surface rough. Cysts present.

On Section.—Tough. Cortex greatly reduced. Demarcation indistinct. Vessels prominent.

HISTOLOGY.—Great overgrowth of fibrous tissue in all positions. Glomeruli atrophied, capsules thickened. Tubules: epithelium degenerated, a little remaining. Arteriosclerosis marked. The degree of fibrosis and degeneration of tubules may vary considerably in different parts of same kidney.

OTHER ORGANS.—Changes in circulatory system constant and marked:—

HEART.—Greatly hypertrophied, especially left ventricle.

ARTERIES.—Thickened.

In the arteriosclerotic form the kidney is about normal size, renal changes as above but not so advanced, with extreme changes in circulatory system.

Pathogenesis.—The renal and circulatory changes are obviously connected, but relationship is unsettled. Theories are:—

1. MECHANICAL.—Renal tissue being reduced, increased blood-pressure is necessary to drive the blood sufficiently fast through what remains. Hypertrophy of heart and arteriosclerosis produce this result.
2. CHEMICAL.—The increase of certain substances in the blood, owing to deficient excretion by the kidneys, causes a rise of blood-pressure. The cardiac hypertrophy and arteriosclerosis are secondary to this rise. In support of this, rise of blood-pressure in parenchymatous nephritis is sometimes found without any apparent arterial thickening.

Symptoms.—

MODE OF ONSET.—

1. INSIDIOUS.—Some initial symptom gradually attracts attention.
2. LATENT.—No symptom until some serious event occurs, e.g., cerebral hæmorrhage. Especially in arteriosclerotic type.

INITIAL SYMPTOMS.—One or more of the following: (1) Dyspepsia; (2) Headache and giddiness; (3) Breathlessness and palpitation; (4) Nocturnal frequency of micturition; (5) Failing sight; (6) General weakness. Worse in general during night and early morning.

APPEARANCE.—Sallow complexion, tired expression, watery eyes; usually thin persons.

GENERAL SYMPTOMS.—

1. URINE.—

Quantity.—Increased. Often 100 oz.

Frequency of micturition.—Especially nocturnal.

Specific gravity.—Persistently low: 1005 to 1012.

Colour.—Pale.

Albumin.—Usually trace only. Temporarily may be absent, especially in morning urine.

Deposit.—A few casts, hyaline or granular.

Urea.—Low percentage.

Chlorides.—Normal.

Chronic Interstitial Nephritis—Symptoms, *continued.*

Renal efficiency tests.—Positive.

Blood urea.—Tends to be increased.

The above are characteristic of granular kidney.

Uric acid often forms considerable (cayenne pepper) deposit, possibly due to scanty pigments and salts.

Hæmaturia occasionally marked and persistent.

In arteriosclerotic type, quantity is less and albumin greater.

With onset of uræmia, frequent but not invariable fall in quantity.

With cardiac failure, quantity usually falls and albumin increases, but paleness and low specific gravity remain.

2. CIRCULATORY SYSTEM (changes important).—

Arteries.—Thickened; may be tortuous and atheromatous.

Pulse.—High tension.

Blood-pressure.—180 to 250 mm. Hg.

Heart.—Apex beat: Displaced down and to the left; impulse forcible. Due to hypertrophy of left ventricle.

Cardiac dullness: Increased, but may be masked by emphysema.

Heart sounds: (1) At mitral area, first sound muffled, or slight systolic murmur (relative insufficiency); (2) At aortic area, second sound greatly accentuated.

Initial symptoms often cardiac: breathlessness and palpitations, frequently worse on lying down, thus preventing sleep.

Cardiac failure occurs with usual symptoms: dilatation of heart, dyspnœa, œdema, increase of albumin and diminution in urine, often fall of blood-pressure.

3. RESPIRATORY SYSTEM.—Bronchitis common; also emphysema. Attacks of dyspnœa, especially nocturnal (cause may be cardiac or uræmic). Cheyne-Stokes breathing often marked towards end.

Respiratory complications are serious: pneumonia or pleurisy. Rare terminations are œdema of lungs or glottis.

4. DIGESTIVE SYSTEM.—Dyspepsia, nausea, and anorexia rarely absent; especially in morning; may be earliest symptom. Tongue furred and breath heavy. Constipation usual: terminal diarrhœa may be intractable. Vomiting may be severe, even without other uræmic symptoms.

5. NERVOUS SYSTEM.—Headache: usually early symptom, and often severe; may resemble migraine. Giddiness. Tingling of extremities. Neuralgias in various sites. Twitchings and cramps of muscles. Cerebral hæmorrhage not infrequent.

Psychical Symptoms.—Irritability and rapid mental fatigue usual. Delusional insanity occasionally.

6. OCULAR SYMPTOMS.—Often earliest complaint: (1) Dimness and failing vision; (2) Amaurosis—often transient, without retinal changes; (3) Conjunctival hæmorrhages. Diplopia rare.

Changes in Fundus.—Albuminuric retinitis; optic neuritis. (*See* OPTIC NEURITIS AND RETINITIS.)

7. ŒDEMA.—Rare, except with cardiac failure, but feet may swell. *Conjunctival œdema* common, 'watery eyes' (the tear which never drops).
8. SKIN.—Dry. Complexion muddy. Eczema and itching common. Urea may deposit on skin in late stages ('urea frost'). Certain of the rarer dermatoses may occur in chronic nephritis, e.g., dermatitis exfoliativa.
9. HÆMORRHAGES.—Common; connected with high tension. Epistaxis (often relieves symptoms); conjunctival; retinal; renal; cerebral. Sputum may be blood-tinged.
10. ANÆMIA.—Secondary anæmia, rarely severe.
11. HEARING.—Noises in ear common; may be transient deafness.

Diagnosis.—

CHARACTERISTICS.—Complaints of headache, weakness, dyspepsia, nocturnal frequency of micturition, or failing vision, associated with arteriosclerosis, albuminuria, and retinal changes. Diagnosis often overlooked, symptoms being referred to individual systems, e.g., gastric.

DIAGNOSIS FROM.—(1) Other causes of frequency of micturition—e.g., enlarged prostate, diabetes; (2) Neurasthenia; (3) Gastritis; (4) Cardiac disease; (5) Chronic bronchitis and asthma; (6) Cerebral lesions (suggested by headache, vomiting, and optic neuritis); (7) Myxœdema; (8) Other causes of uræmia.

Prognosis and Complications.—

DURATION may be many years, with fair health and activity. Condition and reserve power of heart is guide of most importance; urinary changes are less so. With albuminuric retinitis, life rarely exceeds two years.

TERMINATION occurs in following *complications*: (1) Uræmia; (2) Cerebral hæmorrhage; (3) Cardiac failure; (4) Intercurrent infections—pneumonia, pericarditis, pleurisy; (5) Acute exacerbation of nephritis. Possibly spreading œdema of brain. Any one of these symptoms is unfavourable.

Treatment.—No cure is possible. *Indications* are:—

1. To retard progress by removing factors which are contributory and aggravating—viz., overwork, worry, over-eating, alcohol, syphilis, etc. (general treatment).
2. To treat symptoms as they arise.
3. To guard against special sequelæ, cardiac failure, uræmia, and cerebral hæmorrhage.

1. GENERAL TREATMENT.—Regular life without worry or excesses. Moderate exercise. Warm climate in winter. Avoidance of chills. Bowels freely open. Baths to stimulate skin. Plenty of fluids to drink. No alcohol. Vary directions according to patient's position. Undue severity in early stages may cause mental depression. Course at spa will control an unruly patient.

Chronic Interstitial Nephritis—Treatment, *continued.*

Drugs cannot cure. Renal extracts valueless.

DIET.—Moderation important. Light mixed diet. Meat and fish each once daily. Eggs and fruit. Avoid rich foods.

FLUID.—Three to four pints a day. Occasional glass of hot water. If alcohol insisted upon, whisky and soda or light claret best.

BOWELS.—Carlsbad salts on rising. For stronger aperient, pulv. jalapæ co. gr. xv, with pot. tartras acidus gr. xxv, in morning.

2. TREATMENT OF SYMPTOMS.—

a. INCREASED BLOOD-PRESSURE.—Pressure is better high, and if suddenly or greatly lowered, patient feels worse. For reduction of excessive pressure, when straining heart or threatening hæmorrhage:

General means.—Lighter diet. Hot air or water baths. Laxatives.

Drugs.—

℞ Liq. Trinitrini ♏j–ij
t.d.s.

or ℞ Sod. Nitritis gr. ij–iv.
Sod. Nitratis gr. x
t.d.s.

For prolonged administration

℞ Pot. Iod. gr. x
t.d.s.

If pressure too low, give digitalis (cardiac failure).

b. ANÆMIA.—Give iron:—℞ Tinct. Ferri Perchlor. ♏x–xxx
t.d.s.

c. CARDIAC CONDITIONS.—Treat cardiac failure by usual methods. For a few days, tinct. digitalis ♏x, tinct. nux vom. ♏v, t.d.s.; then, if tension high, caffein. citrat. gr. v, t.d.s.

d. GASTRO-INTESTINAL SYMPTOMS.—Loss of appetite and morning nausea usually improve during day. General treatment of light diet, free bowels, and stimulation of skin. Usual treatment of dyspepsia.

e. URÆMIA.—

Chronic Uræmia and Early Symptoms.—Induce sweating by hot-air baths or pack and pilocarpine (inject gr. ⅙ five minutes before bath). Saline purgatives. Nitroglycerin to reduce blood-pressure.

Opium is a valuable and safe drug. Useful for restlessness, insomnia, delirium. Also for dyspnœa and Cheyne-Stokes breathing. Useful also after fits to ward off recurrence.

Coma.—Induce sweating. Open bowels (castor oil, pulv. elaterini co., gr. j–iv, and enema). Venesection: 12 to 20 oz.

Convulsions.—Induce sweating. Chloroform if fits severe.

Venesection: 12 to 20 oz. Inject morphia if fits recur: other sedatives of little avail.

With venesection, intravenous or subcutaneous infusion of sterile physiological saline solution or transfusion of blood. Continuous inhalation of oxygen may be useful. Lumbar puncture has been recommended.

III. SMALL WHITE KIDNEY.

(*Commonly included as a form of chronic parenchymatous nephritis.*)

Morbid Anatomy.—

KIDNEY.—

MACROSCOPIC.—Small (1½ to 3 oz. each). Pale. Capsule thick and adherent. Surface rough.

On Section.—Tough. Cortex reduced, pale and opaque. Demarcation indistinct. Fat in hilus appears to be increased.

HISTOLOGY.—

a. Glomeruli.—Small and atrophied. Glomerulitis. Much fibrous tissue.

b. Tubules.—Little epithelium remains. Fatty degeneration in places.

c. Interstitial Tissue.—Changes prominent. Great relative increase. Arteries thickened.

Symptomatology.—For varieties of clinical course see under Classification of Chronic Nephritis. Small white kidney produces conditions resembling a combination of parenchymatous and interstitial nephritis, or a sequence of interstitial after parenchymatous, or, less commonly, resembling one or the other: this applies to clinical, urinary, and chemical aspects. Cardiovascular and allied changes probably always present in some degree.

Primary small white kidney will produce in young adult the symptoms, cardiovascular and urinary changes, and uremic manifestations characteristic of red granular kidney. Duration usually very short. May simulate cerebral tumour

CHAPTER XCI.

AMYLOID DISEASE.

(*Waxy or Lardaceous Kidney.*)

Amyloid disease is not a form of nephritis, nor is it strictly a renal degeneration, the material being brought by the blood and deposited in the tissues. General amyloid disease usually present, especially spleen and liver.

Amyloid Disease, *continued.*

Nature of Amyloid.—A glycoprotein containing chondroitin-sulphuric acid; this acid is probably brought by blood, and diffusing through walls combines with a local tissue protein. The name 'amyloid' was given from its blue starch-like reaction with iodine.

Occurrence.—Prolonged suppuration and exhaustion—e.g., chronic osteomyelitis, tuberculosis, syphilis. With modern surgical methods is rarely seen.

Morbid Anatomy.—

KIDNEYS.—

MACROSCOPIC.—Pale, usually large, smooth; capsule strips readily. Stellate veins injected.

On Section.—*Translucent waxy appearance.* Large cortex; pyramids deep red; differentiation marked; glomeruli distinct. With Lugol's solution (iodine in potassium iodide solution), walnut-brown points form.

HISTOLOGY.—Changes especially in walls of vessels: structureless and homogeneous. Glomeruli most and earliest affected; next, afferent and efferent vessels; then other vessels. Epithelium not involved, but parenchymatous nephritis often co-exists.

OTHER ORGANS.—Spleen, liver, and intestines affected usually.

Urine.—Amount increased. Pale; low specific gravity.

ALBUMIN. Varies: may be very large amount (often much globulin); sometimes none.

CASTS. Mainly hyaline, rarely numerous.

Symptoms.—*Anæmia.* Often dropsy. *Diarrhœa* common (amyloid intestines). Never uræmia. Also symptoms of primary disease. Arteriosclerosis and cardiac hypertrophy only with co-existing chronic nephritis.

Diagnosis.—Suggested by: (1) Focus of suppuration; (2) Spleen or liver enlarged; (3) Amount of urine large, specific gravity low, much albumin; (4) No arteriosclerosis; (5) Diarrhœa. Fatal ending invariable in cases where recognized.

Treatment.—Of primary condition.

CHAPTER XCII.

PYELITIS.

(Including Pyelonephritis and Pyonephrosis.)

Definition.—Inflammation of the pelvis of the kidney.

PYELONEPHRITIS.—Inflammation involving kidney tissue and pelvis.

PYONEPHROSIS.—Distention of pelvis and calices with purulent fluid.

All conditions in which bacteria (bacilli and cocci) occur in pelvis of kidney are referred to in this section.

Etiology.—By invasion of bacteria. Very rarely, irritation by turpentine, cubebs, or diabetic urine, usually with secondary bacterial infection.

Paths of Infection.—(1) Hæmatogenous; (2) Ascending infection; (3) From surrounding tissues.

1. HÆMATOGENOUS.—Through blood-stream. Normal kidney can eliminate common bacteria harmlessly. Infection occurs in presence of predisposing causes (may be none):—
   a. GENERAL CAUSES.—(i) Specific fevers, especially enteric; (ii) General debility, e.g., severe constipation or anæmia.
   b. LOCAL RENAL CAUSES (conditions disturbing renal function)—(i) Movable kidney (hence pyelitis frequent in women); (ii) Calculus; (iii) Renal operations; (iv) Pressure of neoplasms.
   c. PREGNANCY.—Frequent. By pressure on renal veins or from circulating toxins.
2. ASCENDING INFECTION FROM LOWER URINARY TRACT. Passage along: (1) Peri-ureteral lymphatics; or (2) Lumen of ureter against current. Experimental evidence is advanced for latter possibility.
   PREDISPOSING CAUSES.—*Obstruction to urinary flow* probably always present—e.g., stricture of urethra, enlarged prostate, calculi, neoplasms involving ureteric orifice, diseases of nervous system with retention. 'Catheter fever': from septic instruments. (Dilatation of deep urethra causes vasodilatation and engorgement of kidney.)
3. SPREAD FROM SURROUNDINGS.—Possibly in perinephric abscess.

Morbid Anatomy.—

HÆMATOGENOUS PYELITIS.—Mucous membrane swollen. Later:—
(1) Kidney substance infected—pyelonephritis.
(2) If obstruction, distention of pelvis and calices—pyonephrosis.
(3) Both above may occur, and finally fluid be absorbed and pus inspissated, and be impregnated with lime.

TUBERCULOUS PYELITIS.—Commences at apices of pyramids. Progresses either to: (1) Caseation—frequent; (2) Tuberculous pyonephrosis; or (3) Thickened mucous membrane of pelvis.

ASCENDING PYELITIS ('surgical kidney', 'acute suppurative nephritis').—Numerous abscesses on surface of kidney. Suppuration along pyramids.

Symptoms.—Bacteria in the pelvis of the kidney may be excreted in the urine without causing symptoms, a simple bacilluria, as in enteric fever (*B. coli*, and less often *B. typhosus*, being present in the urine). Conditions in which symptoms occur may be grouped as: (1) Acute pyelitis: (*a*) Hæmatogenous; (*b*) Ascending. (2) Chronic pyelitis. (3) Special forms: (*a*) Fulminating infective nephritis; (*b*) '*Coli* cystitis'.

Pyelitis—Symptoms, *continued.*

1. ACUTE PYELITIS.—

a. HÆMATOGENOUS ORIGIN.—Usually unilateral.

Onset.—Often sudden. Malaise. Pyrexia. Shivering. Pain and tenderness in loin. Frequency and increase of micturition (may occur without cystitis).

Progress.—Symptoms increase. Rigors. Sweats. Swinging pyrexia to 104° F. Kidney tender, but rarely palpable. Such progress indicates pyelonephritis.

Termination and Prognosis.—

(i) Acute symptoms may subside rapidly on early treatment, with frequent recurrence until predisposing cause is removed. In pregnancy, subsides on parturition.

(ii) Acute symptoms diminish and chronic pyelitis develops.

(iii) Pyonephrosis

(iv) Pyelonephritis without operation tends to a fatal end, with symptoms resembling uræmia.

With pregnancy and specific fevers (enteric), resistance to treatment is rare.

b. ASCENDING PYELITIS.—Almost always bilateral.

Onset.—Often obscured by symptoms of primary disease Rigors. Pain and tenderness in loins. Frequency and increase of micturition.

Progress.—Typhoidal state develops, with dry tongue and skin.

Termination.—Predisposing cause often renders treatment valueless; pyelonephritis or pyonephrosis follows, with fatal termination.

2. CHRONIC PYELITIS.—Occurs typically in catheter life. May be sequel of acute pyelitis. Some degree of pyelonephritis present.

Characteristics.—

(a) Pyuria.

(b) Irregular pyrexia. May be intermittent, with *sudden rises to* 103°–104°: may be periodic. Rigors in early stages; cease later.

(c) Septic symptoms marked. wasting, anæmia, nervous irritability.

(d) Tenderness in loin; tumour if pyonephrosis.

Termination by exhaustion or in comatose condition.

3. FULMINATING INFECTIVE NEPHRITIS and 'COLI CYSTITIS' (*see* p. 586).

Complications of Pyelitis.—

1. PYELONEPHRITIS.—Extension of inflammation to renal tissue, especially in ascending pyelitis ('surgical kidneys').

SYMPTOMS.—Severe sepsis, with localizing renal symptoms of pyelitis.

2. PYONEPHROSIS.—Dilatation of pelvis and calices by pus or pus and urine. Results from suppuration in kidney with obstruction to urine.

COMMON CAUSES.—Renal calculus, tuberculosis.

SYMPTOMS.—Sepsis, with renal tumour and signs of obstructive lesion (as in hydronephrosis). Septic absorption and symptoms slighter than pyelonephritis, and there may be no pyrexia.

Diagnosis.—*In all cases of pyuria* the following questions must be considered: (1) Where is the site of infection? (2) What is the predisposing cause? (3) Is condition unilateral or bilateral? (4) What bacteria are present?

SITE OF INFECTION.—

a. CYSTITIS.—May co-exist with pyelitis. In cystitis: pyrexia absent, no sweats or rigors, bladder but no lumbar pains. (Frequency occurs in pyelitis even without cystitis.)

Note.—Pyuria with pyrexia in females is pyelitis; in males, pyelitis or posterior urethritis.

b. POSTERIOR URETHRITIS.—No local signs of pyelitis.

Two-glass Test.—Urine passed into two glasses. In urethritis, 'threads' or turbidity in first, while second is clear. In pyelitis and cystitis, both turbid.

c. PYELITIS.—Pyrexia, sepsis, and localizing signs. General turbidity of urine in 'two-glass test'.

Perinephric abscess may be confused with pyonephrosis; but in former tumour does not move on respiration, and is irregular in shape. Hip often flexed.

PREDISPOSING CAUSE: EXAMINATION OF PATIENT.—

METHODS OF EXAMINATION.—

a. General examination of patient and of urine.

b. Special methods: (i) Radiography: calculi. (ii) Cystoscopy and catheter: strictures, condition of bladder and ureteric orifices (inadvisable in acute stages of pyelitis and cystitis). (iii) Catheterization of ureters; condition of each kidney; ureteric obstructions. Collargol injected into pelvis and radiographed: outlines pelvis and ureter. (iv) Renal efficiency tests for each kidney (*see* p. 567).

NATURE OF BACTERIA PRESENT.—Bacteriological examination of urine. Detects causal bacteria. Catheter specimen essential in female. First morning urine for tubercle bacilli.

Treatment.—(Operation only if definitely indicated.)

1. ACUTE PYELITIS.—

a. MEDICAL.—Rest in bed. Milk diet. No alcohol. Large quantity of fluid (Contrexéville water). Fomentations to tender area. Bowels opened (saline or senna pods).

For drugs, *see* CYSTITIS, p. 598.

✓ No passage of instruments during acute stage, if avoidable.

Pyelitis—Treatment, *continued*

b SURGICAL —Nephrotomy (preferably) or nephrectomy.

Indications — (1) Rapid progress of symptoms (pyelonephritis), (2) Presence of unilateral tumour (pyonephrosis)

Contra indication — Presence of bilateral disease, e g, 'surgical kidneys'

After acute symptoms subside, remove predisposing cause if possible

2 CHRONIC PYELITIS —

a BILATERAL —Mild climate Warm clothes Gentle exercise Liberal diet Contrexéville water Tonics (Surgical treatment contra indicated)

b UNILATERAL Treatment as for acute if resistant then as chronic If resistant and progressing nephrectomy or nephrotomy

Lacunæ —Constitutional symptoms may be allayed even though bacilluria continues especially in secondary and mixed infections

Drugs —*See* CYSTITIS p 598

FULMINATING INFECTIVE NEPHRITIS.

Rare but important Usually in females, unilateral, on right side

Symptoms. *Onset* sudden Pyrexia rigors pain in right loin extending to costal margin rigidity Kidney may be palpable Pulse rapid and feeble

Diagnosis.—From appendicitis pain and rigidity higher

Treatment. With immediate operation and nephrotomy prognosis is good

'COLI PYELITIS', 'COLI CYSTITIS', AND 'COLI BACILLURIA'.

Applied to group of conditions with *B coli* (or bacilli of colon group) in urine, with or without pyuria and symptoms but in absence of the gross local predisposing causes of pyelitis and cystitis

May be no symptoms (simple 'coli bacilluria') If bacilluria without pyuria, any symptoms present probably have other cause Even with pyuria, tuberculosis, etc, must be excluded.

Clinical Types.—

1 NO SYMPTOMS —With pregnancy, enteric fever and predisposing causes, symptoms may arise subsequently

2 PYREXIA, GENERAL MALAISE, AND CHILLS —No genito-urinary symptoms except pyuria

3 ACUTE PYELITIS OR CYSTITIS

4 CHRONIC PYELITIS OR CYSTITIS. May be chronic and mild from onset, or follow acute symptoms

Pyelitis usually present even with symptoms of cystitis only

Severer constitutional symptoms (e g, pyelonephritis) very rare

Predisposing Causes appear to be constipation, hæmorrhoids, intestinal disturbances, and especially pregnancy, but may be absent.

Prognosis.—Final prognosis good, but progress often tedious. Symptoms may subside and bacilluria remain. Great tendency to recurrences.

Occurrence in Children.—Often unrecognized. Commonest under 2 years in females. Probable infection by urethra.

SYMPTOMS.—Two groups :—

(1) General ill-health, often gastric, but no urinary symptoms.
(2) Frequency of micturition, and screaming.

In older children : Chronic condition, with pyrexia, wasting, and ill-health, and no genito-urinary symptoms except pyuria. Often sudden rises to 103°–104°, with normal intervals.

Treatment.—As in PYELITIS and CYSTITIS.

URINE IN INFECTIONS OF URINARY TRACT.

Quantity.—Increased in pyelitis, septic or tuberculous.

Colour.—May be :

CLEAR.—Pus slight and bacilli few, as in early acute pyelitis, and in tuberculosis of kidney and of bladder (until secondary infection) ; at intervals in pyonephrosis or chronic pyelitis.

TURBID.—From pus or bacilli, or phosphates in alkaline urine. Slight turbidity in posterior urethritis.

BLOOD.—May be small amount in any infection.

Albumin.—Necessarily present if pyuria : often trace only.

IN CYSTITIS.—Scanty : agrees with amount of pus

IN PYELITIS.—Often albumin in excess of pus (i.e., pyelonephritis present).

Pus.—Detected by microscope : urine centrifuged if necessary

IN RENAL INFECTIONS.—Pus scanty (urine acid).

IN BLADDER INFECTIONS.—Pus abundant (urine usually alkaline). Forms gray deposit, usually mixed with phosphates.

IN PYELONEPHRITIS.—Maybe casts containing leucocytes.

Epithelial Cells.—In pyelitis, numerous. In cystitis, scanty. No localization possible from shape of cell. (After ureteric catheterization, groups of small round epithelial cells may simulate pus cells.)

Reaction.—Varies with bacteria present :—

B. coli and *B. tuberculosis* : usually acid.

B. proteus and staphylococci : usually alkaline.

In bladder, secondary invasion common with non-pathogenic urea-splitting organisms—e.g., *Micrococcus ureæ*. Hence reaction in cystitis commonly alkaline.

Varieties of Bacteria found.—

✔ *B. COLI* AND COLON GROUP.—Most frequently. Includes

Urine Infections—Varieties of Bacteria found, *continued.*

typical *B. coli*, numerous strains with varying cultural characteristics, and definite strains such as *B. proteus* and less frequently Friedländer's bacillus, *B. pyocyaneus*, and other types.

OCCURRENCE.—Pyelitis, 'coli cystitis', coli bacilluria without symptoms, cystitis, urethritis.

B. TUBERCULOSIS.—

OCCURRENCE: Pyelitis, cystitis.

Colon group and staphylococci often co-exist, and when found, *B. tuberculosis* should be looked for also.

STAPHYLOCOCCUS ALBUS AND *AUREUS.*—

OCCURRENCE.—All sites. Often no symptoms

STREPTOCOCCUS.—Virulent long-chained strains rare. Non-virulent short-chained strains not uncommon.

ENTERIC GROUP.—

OCCURRENCE.—Pyelitis, and in bacilluria without symptoms.

GONOCOCCUS.—

OCCURRENCE.—Urethritis, rarely cystitis.

VARIOUS, without causing symptoms.—Enterococcus common. Rarely, pneumococcus and most pathogenic bacteria.

PARASITES.—*Bilharzia* ova.

Varieties of Bacteria classified according to Lesion.—

PYELITIS.—*B. coli* and colon group. *B. tuberculosis.* Enteric group (in enteric fever). Staphylococcus and streptococcus: uncommon.

CYSTITIS.—*B. coli* and colon group. *B. tuberculosis.* Staphylococcus. Gonococcus. Streptococcus—uncommon.

URETHRITIS.—Gonococcus. Colon group. Staphylococcus.

BACILLURIA WITHOUT SYMPTOMS.—Colon group. Enteric group (in enteric fever).

CHAPTER XCIII.

HYDRONEPHROSIS.

Distention of the kidney by urine as the result of obstruction. The ureters may also be distended, depending on site of obstruction.

Etiology.—Causal obstruction must be incomplete, intermittent, or gradual, since sudden complete obstruction results in renal atrophy without distention. May be unilateral or bilateral. The causes may be (1) Congenital; (2) Acquired.

1. CONGENITAL.—Ureter twisted or contracted, or inserted into pelvis of kidney at acute angle or in abnormal position; these include many of the large tumours. Constriction of ureter by abnormal branch of renal artery.

Congenital bilateral hydronephrosis usually occurs with other abnormalities—e.g., club-foot—and is fatal in a few days; frequently due to imperforate urethra.

2. ACQUIRED.—

a. IN LUMEN OF URETER.—(i) Calculus obstructing ureter, or causing ulceration with subsequent stricture; most common cause. (ii) Tumours of bladder.

b COMPRESSION OF URETER.—(i) Movable kidney kinking ureter (intermittent form). (ii) Pressure of tumours of ovaries or uterus. (iii) Contraction of cellular tissue following pelvic inflammation. More rarely: bands of fibrous tissue, enlarged lymphatic glands, various tumours and neoplasms

c. BILATERAL HYDRONEPHROSIS (rarely palpable)—Phimosis. Stricture of urethra. Enlarged prostate. Tumours of bladder (may cause unilateral hydronephrosis).

EFFECTS ON KIDNEY.—Pelvis and calices enormously distended. Kidney finally becomes sac of fluid, lobulated by persistence of the interlobular septa. The renal tissue distends and atrophies, but a small layer is usually present even in advanced cases. The fluid contains salts, a trace of urea, and occasionally of albumin. There may be adhesions to other organs and compression of colon Palpable tumour is often absent, but it may be enormous The opposite kidney may enlarge in compensation.

SEX.—Twice as frequent in women as men, owing to association with movable kidney and pelvic diseases.

AGE.—May occur at any age from congenital causes.

Symptoms.—

TUMOUR.—May occupy most of abdomen; surface smooth or lobulated; tense or elastic, or may fluctuate; in general, resembles renal tumour—viz., bulges into flank, with colon in front; dull to percussion; often painless.

No characteristic symptoms in absence of tumour; there may be obscure abdominal pains, pain in back, with frequency or diminution of urine.

INTERMITTENT HYDRONEPHROSIS — Tumour disappears, with large discharge of urine, and then refills. Caused by movable kidney kinking ureter.

Diagnosis.—*Intermittency*, when occurring, is diagnostic. *Catheterization of ureters* and X rays often conclusive.

1. LARGE TUMOUR.—From *ovarian tumour*: Latter is more mobile, tends to enlarge upwards rather than into flank, colon and intestines behind, uterus often displaced up and to side. Diagnosis difficult Occasionally confused with ascites.

2. MODERATE TUMOUR.—From: (a) *Pyonephrosis*: Pyuria and signs of sepsis. (b) *Perinephric abscess*: Rapid onset, painful, signs of sepsis. (c) *Hydatid cyst*. Also *gall-bladder, cystic kidneys, tumours of kidney, Riedel's lobe.*

Hydronephrosis, *continued.*

Prognosis.—Depends partly upon cause.

1. UNILATERAL.—Often no symptoms. Size may cause discomfort.

   COMPLICATIONS.—(a) Pyonephrosis from suppuration; (b) Blockage of sound ureter by calculus, and hence uræmia; (c) Rupture of sac into peritoneum.

2. BILATERAL.—Uræmia not uncommon.

Treatment.—No medical treatment. Operate for increasing size or symptoms.

OPERATION.—Aims at removing cause—e.g., by fixation of movable kidney. If sac thin, do nephrotomy and drain, or nephrectomy, but save kidney if possible. In nephrotomy, examine for stone in ureter. Before nephrectomy, consider excretion of opposite kidney by phenolsulphonephthalein etc.

CHAPTER XCIV.

NEPHROLITHIASIS.

(*Renal Calculus*)

The formation of concretions in the kidney or its pelvis.

Occurrence of Calculi and Concretions.

1. IN THE KIDNEY SUBSTANCE—
   (a) In new-born children, uric acid particles, at apices of pyramids. Passage may cause crying and priapism.
   (b) In gouty and other persons, urates occur at apices of pyramids.
   (c) In old people, white deposits of calcium carbonate may be found in the pyramids.

   Such deposits in adults cause no symptoms, and all are of little importance.

2. IN PELVIS AND CALICES—
   (a) Renal sand or 'gravel'. Small particles of uric acid passing into urine and forming red 'cayenne-pepper' deposit.
   (b) Small stones, single or multiple. Passage causes renal colic.
   (c) Single dendritic calculus. May occupy entire pelvis, forming accurate mould of all depressions.

Characters of Renal Calculi.—Calculi causing renal colic are usually ¼ to ½ inch in diameter; often pigmented; if multiple, may be faceted. When removed from ureters, are usually either oblong or 'mulberry' calculi.

ON FRACTURE, surface often shows a cortex with concentric

rings, and a nucleus The composition of nucleus may differ from cortex, and the rings may be of different or similar composition.

Chemical Composition of Urinary Calculi.—

1. CALCIUM OXALATE WITH VARYING AMOUNT OF CALCIUM PHOSPHATE - Commonest calculus in kidney and ureter Smooth surface, pale, often oblong
2. CALCIUM OXALATE (pure) —Reddish colour Irregular surface. 'Mulberry' shape Very hard and painful
3. TRIPLE PHOSPHATE (ammonio-magnesium phosphate) Common in bladder Rare elsewhere Large, soft, and very friable
4. URIC ACID -Very rare in kidney and ureter, more common in bladder Smooth brown fairly hard Does not show in radiograph. A trace of uric acid is not uncommon in oxalate or phosphate stones
5. AMMONIUM URATE —Hard brown colour
6. MIXED COMPOSITION —Uric acid nucleus, with cortex of other composition Calcium phosphate may occur in other calculi, or with an outer layer of triple phosphate

Rare calculi are —

CYSTINE Large, soft, and 'soapy' to touch Crystallizes from alcohol in hexagonal plates (*See* CYSTINURIA p 562)

XANTHINE —Burns without a flame

CALCIUM CARBONATE

UROSTEALITH.

Mode of Formation of Calculi.—Causes not yet ascertained. Factors of importance in formation of common renal and ureteric calculi are high acidity, low pigmentation, high concentration of solids, and possibly inflammation of mucous membrane Acidity is probably chief factor

Etiology.—

AGE —Usually after middle life

SEX —Males twice as common as females

PREDISPOSING FACTORS —Excessive diet, possibly sedentary life (common in fat men) Probably certain drinking waters: thus is specially common in certain localities, e g, Norfolk.

THE TWO KIDNEYS EQUALLY LIABLE —Occurs on both sides in nearly one sixth of cases

Symptoms.—*Acute renal colic* occurs when a calculus tries to enter or move along a ureter which impedes its progress Calculus may be anywhere in ureter, but most commonly at (1) Exit from pelvis, (2) When ureter crosses iliac vessels, (3) Entry in bladder

ATTACK OF ACUTE RENAL COLIC —

(1) Sudden onset of agonizing pain. May be after exertion or jolting, or without apparent exciting cause

Nephrolithiasis—Symptoms, *continued.*

(2) Pain commences in kidney region and radiates down ureter to groin, inner side of thigh, and to testis or labium on same side: testis retracted and tender. No relief in any position.

(3) Micturition frequent, painful, and bloody (bladder irritable).

(4) Nausea and often vomiting. Pulse rapid and feeble. Perspiration. Collapse. No rise of temperature.

DURATION.—One to many hours. Often subsides suddenly.

PAIN.—Often radiates from two renal points on abdomen and back. Pain extremely acute here, and also *dull ache* after spasms.

URINE.—May be none during attack. Sometimes much clear urine, possibly from other kidney.

AFTER ACUTE PAIN SUBSIDES.—

(1) Dull ache in back over kidney. Often localized to renal points, tender on pressure.

(2) Hæmaturia; rarely profuse; may persist several days. Often some pyuria.

(3) The calculus may be passed through urethra.

If other kidney diseased, complete suppression of urine (anuria) and uræmia sometimes occur.

A small rough oxalate stone produces more pain and hæmaturia than larger smooth calculus.

CALCULI REMAINING IN PELVIS may cause:

(1) No symptoms—e.g., large dendritic or small fixed calculi.

(2) Acute renal colic—possibly by entering ureter

(3) Commonly some of following symptoms:—

a. Constant pain in renal region; of varying intensity, may radiate. Sometimes referred to opposite side

b. Hæmaturia for irregular periods, rarely profuse, may be only tinge, relieved by rest Frequency of micturition Pyuria.

c. Pyelitis: From secondary bacterial infection (pain in back; pyrexia, 103°–104°; chills and sweats). Also pyonephrosis and pyelonephritis.

Dyspepsia and weakness from constant pain

Pain may simulate sciatica, or be referred to heel or foot.

(Continual spasm of muscles occasionally causes lateral inclination of trunk)

Renal sand may cause dull aching and frequency of micturition.

Diagnosis.—Depends upon:—

1. SYMPTOMS.—With acute renal colic, often simple: (1) Direction of radiation; (2) Hæmaturia.
2. PHYSICAL EXAMINATION.—During acute attack negative. Subsequently often tenderness of renal points. Large calculus very rarely palpable through abdomen (in thin people). When low in ureter, often palpable through vagina or rectum.
3. RADIOGRAPHY.—Usually conclusive, but uric-acid calculus throws no shadow. Simulation of calculi by calcareous glands in line of ureter.

4. CYSTOSCOPY.—Excretion of urine and condition of ureter.
5. CATHETERIZATION OF URETER.—Excludes calcareous glands. Urine from each ureter collected and examined.

Differential Diagnosis.—

1. INTESTINAL COLIC.—Distribution of pain less definite.
2. MOVABLE KIDNEY.—Dietl's crises may simulate renal colic. Kidney palpable, usually right side and in women.
3. BILIARY COLIC.—Pain radiates to shoulder. Jaundice may follow.
4. RENAL TUBERCULOSIS.—Wasting. Hæmaturia unconnected with pain. Tubercle bacilli in urine.
5. CALCULUS IN BLADDER.—From ureteric calculus often clinically difficult. Urine often alkaline. Radiography and cystoscopy.

Treatment.—

1. ACUTE RENAL COLIC.
   Indication is to ease pain. Hypodermic injection of morphia gr. ¼ and atropine gr. $\frac{1}{100}$. Hot drinks of lemonade. Hot poultices to site of pain. Rest in bed until hæmaturia ceases.
2. BETWEEN ATTACKS.—(*a*) Medical; (*b*) Surgical.
   a. MEDICAL TREATMENT.—No drug or other treatment can dissolve a calculus.
   Indications.—(i) Keep urine in condition unfavourable for deposition—viz., low acidity (for oxalates and uric acid); (ii) Diminish excretion of such substances. Regular life. Open bowels. No sudden exertion.
   Diet.—Rich in vegetables (vegetable acids are excreted as carbonates). Avoid food with much purins and oxalates, viz.: (i) Purins: rich meats, e.g., sweetbread; (ii) Oxalates: rhubarb, spinach, strawberries, and tomatoes. Whey, a teacupful three times a day, aids excretion.
   Fluid.—Daily large quantity of minerals, especially lithia (but action mainly due to water).
   Drugs.—Reduce acidity with pot. citras; acidity highest in morning urine (no excretion of HCl in stomach); give ʒj at night and gr. xx in morning. Test urine, and aim at neutrality, to prevent deposition of phosphates (Langdon Brown).
   Spa.—Contrexéville has great reputation for cures.
   b. SURGICAL TREATMENT.—
   Indications.—Attacks of renal colic and aching pains. Chronic pyuria. Evidence of pyelitis and infections of kidney.
   Contra-indications.—Calculi in fat people. Condition of other kidney must be ascertained.
   Calculi often recur, and, after operation, medical treatment must be followed.

CHAPTER XCV.

TUMOURS OF THE KIDNEY. CYSTIC DISEASE OF THE KIDNEY. PERINEPHRIC ABSCESS.

TUMOURS OF THE KIDNEY.

BENIGN TUMOURS.

Rare: of little importance. Fibroma—most common. Adenoma—may be benign or malignant. Lipoma. Lymphadenoma. Tumours of pelvis: (1) Angioma of pelvis—may cause persistent, even fatal, hæmaturia; (2) Papilloma—may grow to large size.

MALIGNANT TUMOURS.

Primary or secondary.

General Description.—

VARIETIES OF PRIMARY TUMOUR.—

1. CARCINOMA.—Very rare.
2. SARCOMA.—More common. Usually spindle-celled. Rhabdomyoma (striped muscle fibres) very rarely: in infants, early death.
3. EMBRYONIC TUMOURS.—In children. Various embryonic tissues present. May grow rapidly to large size.
4. HYPERNEPHROMA (Grawitz's tumour).—

Origin.—Formerly believed to be suprarenal 'rest', since (*a*) situated just below renal capsule, (*b*) at upper pole of kidney, (*c*) resembles suprarenal cortex and tumours, (*d*) does not infiltrate kidney. Now considered to be a true renal adenoma or adeno-lipoma. (Cortex of kidney and suprarenal are related embryologically, explaining similarity.)

Morbid Anatomy.—(*a*) Macroscopic: *Yellow opaque areas* (characteristic); hæmorrhage and cysts common. (*b*) Histology: Polygonal cells with alveolar arrangement, in circular columns; general resemblance to suprarenal cortex; much fat and glycogen.

Age.—At any age, usually children or at 40 to 50 years.

Unilateral. Grows rapidly and to large size. May invade and grow along renal vein and produce emboli; rarely into renal pelvis. Metastases: in lung, and not infrequent in liver, bones, and also other sites.

IN CHILDREN.—Retroperitoneal and renal sarcomata are commonest *large* abdominal tumours.

CHARACTERISTICS.—Age: 3 to 4 years. Early symptoms: (1) Wasting and anæmia; (2) Abdominal enlargement.

Hæmaturia often absent. Physical signs Unilateral tumours, often very large

IN ADULTS.—

SYMPTOMS —

1 *Hæmaturia* —Usually is earliest symptom Often intermittent

2 *Pain* Variable, may be none, or dragging in loin, severe on passage of clots

3 *Wasting* —Usually rapid

PHYSICAL SIGNS —

Tumour (*a*) Tends to fill flank and then extend towards mid-line, (*b*) Often movable, but may be fixed, (*c*) No movement on respiration, (*d*) Crossed by colonic resonance, (*e*) Resonant area between tumour and liver or ribs—but absent if tumour very large May fill half abdomen

Varicocele on left side occasionally tumour penetrating renal vein may obstruct left spermatic vein pulmonary embolism may occur

Diagnosis.—*Characteristics* are (1) Hæmaturia, (2) Pain, (3) Tumour Diagnose —

1 By character of tumour, from spleen or liver if very movable, resembles cancer of ovary

2 Hematuria from other causes

Cystoscopy and catheterization of ureters determines affected side Functional activity of diseased kidney is diminished The occurrence of portions of neoplasm in the urine is a pathological curiosity

Treatment.—Nephrectomy Prognosis very bad

CYSTIC DISEASE OF THE KIDNEY.

POLYCYSTIC KIDNEYS: CONGENITAL CYSTIC KIDNEYS.

Etiology.—May occur in fœtus, obstructing labour Symptoms most commonly at 40 to 50 years Probably all congenital May be undiagnosed and found post mortem

Morbid Anatomy.—*Bilateral*, but one side usually larger than other Often very large Surface irregular from protruding cysts

ON SECTION —Numerous cysts, contents clear or turbid fluid, containing albumin and phosphates but no urea, cysts open into each other, but no communication with pelvis or calices *No obstruction in ureters* Remnants of renal tissue may be visible, but, even if absent, microscope often shows unexpected amount

Rarely Liver and spleen also cystic.

Pathogenesis.—(1) Generally ascribed to remnants of mesonephros (Wolffian body) included in metanephros or true kidney. In infants, there may also be imperforate anus (2) Less probably an endothelioma of kidney. No obstruction of ureters present.

Cystic Disease of the Kidney, *continued.*

Symptoms.—

1. Bilateral renal tumour: may increase in size.
2. Hæmaturia.
3. Symptoms and signs of chronic interstitial nephritis thickened arteries, hypertrophy of heart, urinary changes

Termination.—As in chronic nephritis—uræmia, etc. Rarely, rupture of cyst and perinephric abscess.

Treatment.—Palliative. Operation useless.

VARIOUS CYSTS.

Echinococcus Cysts.

Solitary Cysts.

Small Cysts in Chronic Nephritis, from obstruction to tubules.

Diffuse Cystic Disease of kidneys, liver, spleen, and sometimes thyroid. Cysts small and not numerous. May be related to congenital cystic disease. Very rare.

PERINEPHRIC ABSCESS.

Suppuration of connective tissue round kidney.

Etiology.—Infection commonly through blood Occasional local causes are trauma, or extension from kidney, appendix, or spine *B. coli* most common organism; pneumococcus not infrequent A chronic perinephritis, with extensive fibrosis, also occurs.

Symptoms.—*Onset* often insidious, with signs of sepsis pyrexia and sweating. *Pain* in lumbar region variable. Hip joint often flexed. May be pyuria In lumbar region, skin may be red and œdematous: tender on pressure. *Tumour in loin*: irregular shape, no movement on respiration or palpation.

Diagnosis.—From:—

RENAL TUMOURS.—Usually movable. Hæmaturia

TUBERCULOUS HIP.—Joint swollen; *resists rotation* as well as extension.

Treatment.—Drainage.

CHAPTER XCVI.

CYSTITIS.

Inflammation of bladder due to bacterial infection.

Origin and Effects of Bacteria in Bladder.—Bacteria can pass through bladder harmlessly. Predisposing causes lead to infection. In absence of these, apparent cystitis (e.g., 'coli cystitis') is usually associated with pyelitis or, less often, posterior urethritis. (*See* PYELITIS.)

PATHS OF INFECTION.—

1. DESCENDING.—From kidney—pyelitis or direct from blood-stream.
2. ASCENDING.—From lower urinary tract—by instruments or previous inflammation.
3. BY EXTENSION.—From surrounding structures, *rarely*.

CAUSES PREDISPOSING TO INFECTION.—

COMMON.—Stricture of urethra (gonorrhœa); enlarged prostate; calculus or neoplasm of bladder; diseases of nervous system; diabetes; passage of septic instruments; injuries

RARE.—Irritants in urine—e.g., turpentine, cubebs; parasites—*Bilharzia*; foreign bodies.

Cold, debility, alcohol may explain certain cases.

Clinical Types of Cystitis. (1) Acute; (2) Chronic; (3) Tuberculous (*see* TUBERCULOSIS OF THE BLADDER, p. 600).

Etiological Types.—(1) 'Coli cystitis' and pyogenic organisms; (2) Gonorrhœal; (3) Tuberculous.

Morbid Anatomy.—

IN ACUTE FORM.—Mucous membrane swollen, often covered with mucus, and with numerous ecchymoses. Neighbourhood of trigone and ureters earliest and most affected.

IN CHRONIC FORM. Fibrosis often leads to contracted bladder. Irregular ulceration and sloughing of mucosa results in thinning of wall, with pouches and trabeculæ

Symptoms.—

ACUTE CYSTITIS.—(1) Pain above pubes and in perineum; (2) Frequent desire to micturate; (3) Agonizing pain in bladder and end of penis on micturition; (4) Pyuria. No pyrexia, sweats, or rigors.

CHRONIC CYSTITIS.—Similar but less marked symptoms. Usually secondary to acute cystitis, but onset may be gradual—e.g., in retroverted gravid uterus.

Diagnosis, Diagnostic Methods, Condition of Urine, Bacteriology.—*See* PYELITIS, pp. 582–8.

Treatment.—

1. ACUTE CYSTITIS.—Bed. Milk diet. No alcohol. Bowels opened with mild aperients (salts, senna pods). Daily enema. Suprapubic fomentations. Large quantity of fluids (Contrexéville water). Hip-baths, two or three daily for 30 minutes, very hot. Drugs (*see below*). No instruments during acute stage.

   WHEN SUBSIDING.—Bladder washes.

2. CHRONIC CYSTITIS.—

   Remove any predisposing cause if possible.

   General treatment: warm climate; mild exercise; liberal diet; large quantity of fluid (Contrexéville water).

   Drugs. Vaccine treatment.

   Bladder washes: Janet's method.

   Suprapubic cystotomy: if other measures fail.

Cystitis—Treatment, *continued.*

DRUGS.—

1. *Urine acid.* Alkalis and sedatives:—

℞ Pot. Citratis — Tinct. Hyoscyami ♏x l
Pot. Bicarb. āā gr. x — Inf. Buchu ad ℥j
t.d.s.

2. *Urine alkaline*:—

℞ Hexaminæ gr. x — Aq. Menth. Pip. ad ℥j
Acid Sod. Phosphat. ʒss
t.d.s.

VACCINES.—In 'mixed' infections, pyogenic organisms with *B. tuberculosis* or gonococci, vaccines are sometimes of value allaying constitutional symptoms.

BLADDER WASHES.—

Posterior irrigation, by Janet's hydrostatic method. Of special value in gonorrhœal forms. Solution of saturated boric acid, or silver nitrate gr. ij to iv to the pint.

Instillations later.—Catheter passed 1 inch behind compressor urethræ; injection by syringe of silver nitrate solution ʒss—strength gr. v to the *ounce*

CHAPTER XCVII.

TUBERCULOSIS OF URINARY TRACT.

I. TUBERCULOSIS OF THE KIDNEYS.

Etiology.—

MODE OF ORIGIN.—In fatal phthisis there may be foci in kidneys, and in generalized tuberculosis often some miliary tubercles of no clinical importance. *Clinical renal tuberculosis* may have origin

1. THROUGH BLOOD-STREAM. Often secondary to slight pulmonary focus or chronic bronchial gland. Rarely is primary.
2. THROUGH ASCENDING INFECTION.—From bladder, prostate, epididymis, etc.
3. Direct spread from tuberculous vertebra has occurred.

AGE.—Commonest, 20 to 30 years.

SEX.—Males commoner than females.

Morbid Anatomy.—Surface of kidney may appear normal.

IN BLOOD INFECTIONS.—

(a) Commences at base of pyramid in upper or lower pole. Caseous nodule forms, tends to soften and to burst into pelvis; hence ragged cavity; pyelitis may follow; tuberculosis tends to spread down ureter. Nodules may be multiple, some being caseous and some ruptured.

(b) Less commonly, commences in pelvis and attacks pyramids; or ureter chiefly affected, being infiltrated and thick.

IN ASCENDING INFECTIONS.—Lower pole usually attacked, spreads inwards from apex of pyramid. Ureter thickened.

Nodules spread by formation of surrounding miliary tubercles, which grow and coalesce.

Intervening renal tissue may be healthy, fibrosed, or occasionally show changes of nephritis.

Nearly always attacks one kidney first, affection of other following much later.

PROGRESS OF DISEASE.—Many months or several years before kidney is destroyed.

RESULTS may be:—

1. Kidney forms sac containing thick putty-like material.
2. Fibrosis marked: pelvis and capsule thickened, scattered caseous nodules.
3. Pyonephrosis: from blocking of ureter with tuberculous growth.
4. Ureter thickened and ulcerated.

Other changes may be:—

Secondary infection with pyogenic bacteria.

Spread down ureter to bladder, vesiculæ seminales, testes, prostate: in late case with widespread disease, primary focus often doubtful

Glands in hilus enlarged.

Adhesions to surrounding structures.

Symptoms.—

1. FREQUENCY OF MICTURITION.—Night and day. Most frequent early symptom. Due to: (a) Polyuria; (b) Non-tuberculous inflammation of trigone; and partly to reflex irritation.
2. PAIN.—May be: (a) Renal—dull ache in loin: frequent. (b) Bladder—at end of penis: from passage of tuberculous matter, even before bladder affected.
3. PYURIA. May be absent at times if ureter blocked.
4. HÆMATURIA. Slight, except early: rarely, profuse from pelvis.

In unilateral disease.—General health fair.

In bilateral disease.—Constitutional symptoms more marked, irregular pyrexia, rigors, loss of weight. Phthisis common.

Physical Signs. Often no local renal signs. Occasionally: (a) Tenderness on pressure. (b) Kidney palpable—unusual: large tumour rare except with pyonephrosis. Tuberculous nodules may be palpable in testes, vesiculæ, or ureters (per rectum).

Urine.—*Amount* increased; *specific gravity* low; *colour* pale; *reaction* acid; *pus* varies, often settles in white layer, leaving urine clear; *blood* usually absent; *casts* rare. Tubercle bacilli may be present.

Diagnosis.—Questions arising are: (1) Is renal tuberculosis present? (2) Which kidney is affected? (3) What is condition of other kidney? (4) What is condition of genito-urinary tract?

Tuberculosis of the Kidneys—Diagnosis, *continued.*

PRESENCE OF RENAL TUBERCULOSIS.—

a. SUGGESTIVE SYMPTOMS.—(i) Frequency of micturition without arteriosclerosis; (ii) Pyuria; (iii) Tuberculosis of testis, vesiculæ seminales, prostate, or lungs.

b TUBERCLE BACILLI IN URINE.—(Tubercle bacilli may possibly occur in urine without renal tuberculosis; hence bacilli, in absence of pus and symptoms, would not *prove* renal tuberculosis).

CONDITION OF EACH KIDNEY.—

a. CYSTOSCOPY.—(i) Pus issuing from ureter may be visible; (ii) If ureter involved, ureteric orifice is red, dilated, and is displaced outwards by contraction of fibrous tissue in ureter; (iii) There may be tubercles locally in bladder

b. CATHETERIZATION OF URETERS.—Urine examined from each kidney: injection of indigo-carmine or phenol-sulphonephthalein previous to catheterization

c X RAYS.—Tuberculosis gives indefinite shadow.

EXAMINATION OF TESTIS, PROSTATE, VESICULÆ SEMINALES, AND URETER.—Also of lungs.

IN RENAL CALCULUS.—(1) Pain persists with rest, (2) Hæmaturia with attack of colic, (3) Definite shadow in X ray.

Treatment.—

OPERATION.—Renal tuberculosis never heals, and operation is the only cure.

INDICATIONS FOR OPERATION.—(1) Unilateral disease, (2) Pyonephrosis, even with some disease in other kidney.

CONTRA-INDICATIONS.—(1) Bilateral disease; (2) Tuberculosis elsewhere *causing constitutional symptoms*

WHEN OPERATION IS CONTRA-INDICATED.—Treat as tuberculous cystitis (*see below*)

TUBERCULIN TREATMENT.—Subsequent to, but not replacing, operation, and in inoperable cases

Prognosis after Operation.—If bladder unaffected, and no disease in epididymis, vas, or prostate, prognosis is good, but other kidney may become infected at any interval subsequently

II. TUBERCULOSIS OF THE BLADDER.

(*Tuberculous Cystitis*)

Most common in young males. *Always secondary* to tuberculosis of kidney, epididymis, or possibly prostate. Spreads along lymphatics of ureter or vas, which become thickened.

Morbid Anatomy.—

EARLY.—Red tubercles form, caseate, and form ulcers covered with slough and surrounded with gray tubercles.

SITE.—(1) *Around ureteric orifice*, secondary to kidney; (2) Outer side of ureter, secondary to epididymis.

LATER.—*Fibrosis causes extreme contraction of bladder.* Perforation very rare. Secondary infection may occur, ulceration marked, and phosphatic deposits.

Symptoms.—(1) Frequency of micturition, especially at night. (2) Pain in bladder and end of penis on micturition. (3) Urine pale, acid, clear or purulent; blood not common.

Diagnosis.—

METHODS.—(1) Examination of urine for tubercle bacilli and pyogenic organisms; (2) Condition of epididymis and vas, and of kidneys and ureter; (3) Cystoscopy.

DIAGNOSIS FROM.—Other causes of cystitis, and from pyelitis, calculi, neoplasms, *Bilharzia.*

Prognosis.—Grave.

1. When primary focus is removed, small lesion occasionally heals, but often relapses.
2. Progressive, commonly; bladder contracts; finally back-
3. Rapid extension and complications: fistulæ, secondary bladder infection, tuberculous peritonitis, general tuberculosis. pressure to kidneys, and uræmia.

Treatment.—

OPERATION.—Removal of primary focus, kidney or testis, with ureter or vas. (Exclude bilateral renal tuberculosis previously.)

General measures to maintain health.

Operations on bladder and all local treatment of bladder valueless. Drugs useless.

TUBERCULIN TREATMENT.—Should be used. Avoid causing reaction: resulting rise of temperature should not exceed 99° F. Begin with not more than 0·0001 c.c. tuberculin R. Injections fortnightly. Increase slowly. If reaction occurs, re-commence with smaller dose. Continue many months.

VACCINE TREATMENT.—If secondary infection with pyogenic organisms occurs.

III. TUBERCULOSIS OF THE PROSTATE.

Frequent in genito-urinary tuberculosis, but rarely if ever primary.

Symptoms.—(1) Pain and frequency of micturition; (2) Pain on defæcation; (3) Extreme pain on catheterization. Symptoms may be latent, discovered on routine examination in genito-urinary tuberculosis.

Physical Signs.—*Per rectum*: nodules in prostate; fairly hard, may be unilateral. Examine epididymis, vas, etc., for signs of tubercle. Massage prostate and examine secretion for tubercle bacilli and pus.

Diagnosis.—Usually by other genito-urinary tuberculosis. (1) From gonorrhœa: presence and examination of discharge, epididymitis. (2) From cancer: neoplasm much harder

Treatment.—Remove primary disease by operation. Tuberculin treatment. Sedatives for bladder.

IV. TUBERCULOSIS OF THE TESTIS AND EPIDIDYMIS.

Etiology.—(1) *Primary*: not uncommon. (2) *Secondary* to genito-urinary or other focus. Presence occasionally diagnoses an obscure tuberculous peritonitis.

AGE.—May occur in infants, when prognosis is bad owing to generalization.

INJURY.—Often calls attention to nodule; no definite proof of causation.

Morbid Anatomy.—Commences in *globus major* of epididymis, nodule forms; spreads through epididymis, which becomes irregularly thickened round testis; then along vas to remainder of genito-urinary tract. Testis often free until late stage. Caseation and softening may produce cold abscess, which becomes adherent to scrotum and bursts.

Symptoms.—Usually no symptom until attention attracted by pain on casual pressure; testicular sensation remains.

SIGNS.—Nodule in globus major, unilateral at onset; chronic, progress slow. Later, entire epididymis becomes thickened, hard, and irregular. Small hydrocele common. Spread along vas gives sensation of 'beading'. Testis rarely much enlarged.

Rarely: Acute onset with pain.

Diagnosis.—From:—

1. GUMMA.—Affects testis. Testicular sensation diminishes. Improves with antisyphilitic treatment. Wassermann reaction positive.
2. GONORRHŒA.—Affects *globus minor* initially. Presence and history of discharge.
3. NEOPLASM.—Affects testis and epididymis. Rapid growth.

Treatment.—Not uncommonly heals under general and tuberculin treatment.

TUBERCULIN TREATMENT.—Results may be good in this form of tuberculosis. Accessibility of testis and vas renders it easy and safe to watch disease during treatment.

OPERATION.—

INDICATIONS.—Condition spreading under treatment; vas affected. Early stage, partial epididymectomy. Later, remove testis and vas so far as possible.

Tuberculin treatment after operation.

CONTRA-INDICATION TO OPERATION.—Extensive disease elsewhere.

Section VIII.—DISEASES OF THE BLOOD.

CHAPTER XCVIII.

ANÆMIA.

I. NORMAL BLOOD.

In a healthy adult male, age 20 to 40 years, the following figures are normal:—

Red cells (erythrocytes) ..	5,500,000 to 7,000,000 per c mm. (usually 6,000,000 to 6,500,000 per c.mm.)
Hæmoglobin ..	95 to 100 per cent
Colour index ..	0·8 to 0·9
White cells (leucocytes)	5000 to 8000

Differential Count of White Cells:—

Polynuclear neutrophils or finely granular oxyphils	60 to 72 per cent
Eosinophils or coarsely granular oxyphils	2 to 3 per cent
Mast cells or coarsely granular basophils ..	0·1 to 0·5 per cent
Large hyalines or large mononuclears	4 to 8 per cent
Small lymphocytes .. 15 to 25 per cent Large lymphocytes .. 5 to 10 per cent	20 to 35 per cent

The *Colour Index* is a measure of the amount of hæmoglobin contained in each red cell compared with the normal amount. It is calculated thus:—

$$\frac{\text{Hæmoglobin per cent}}{100} \div \frac{\text{Number of red cells}}{5{,}000{,}000}$$

(For comparison, 5,000,000 per c.mm. is used as standard.)

The number of red cells is commonly lower in females, and also falls slightly from the age of 40 to 45 onwards.

II. GENERAL CONSIDERATION OF ANÆMIA.

Anæmia means a reduction in the erythrocytes or hæmoglobin of the blood. Also applied to the symptoms resulting.

Causes.—(1) Removal of blood; (2) Destruction of cells in the body (hæmolysis); (3) Deficient or defective formation.

Classification.—Anæmias are divided into: (1) *Primary*—no recognized cause, and (2) *Secondary*, or symptomatic—a recognizable cause present.

1. PRIMARY ANÆMIA.—
   - *a.* Chlorosis.
   - *b.* Pernicious anæmia.
   - *c.* Aplastic anæmia. Rare.
   - Doubtful, leukanæmia.

Anæmia, General Consideration of —Classification, *continued*

2. SECONDARY ANÆMIA —Principal causes are :—
   (a) HÆMORRHAGE —*Conditions opening large vessels* Trauma. Gastric, duodenal, or typhoid ulcers. Hæmorrhoids. Uterine fibroid. Post- or ante-partum hæmorrhage. Hæmoptysis Tubal pregnancy. Aneurysm Hæmophilia
   (b) INANITION —*Interference with the reception, absorption, and utilization of food.* Especially. Starvation, tuberculosis and cancer (markedly of stomach and œsophagus), prolonged lactation Also chronic sepsis, nephritis, chronic dyspepsia, and septic teeth
   (c) CERTAIN INFECTIONS —Especially malaria, typhoid, diphtheria, syphilis, rheumatic fever To some degree in most specific fevers
   (d) BLOOD DISEASES
   (e) VARIOUS CONDITIONS AFFECTING LYMPHATIC GLANDS AND SPLEEN e g, splenic anæmia, lymphadenoma
   (f) INTOXICATIONS AND DRUGS e g, lead, trinitrotoluene, potassium chlorate
   (g) ANIMAL PARASITES —*Ankylostoma, Dibothriocephalus latus*

In secondary anæmia, the blood changes are common to most forms, but cannot be employed for classification Among difficulties in classification are (1) In chlorosis, changes closely resemble secondary anæmias, (2) In *Dibothriocephalus latus* infection changes may be identical with pernicious anæmia; (3) Leukæmia may be classified as primary and secondary, and produce either type of change, (4) Aplastic anæmia may result from trinitrotoluene and some other forms of poisoning.

General Diagnosis of Anæmia.—

1 INSPECTION —Must always be confirmed by examination of blood Colour of cheeks no guide fever, excitement, sunburn, natural complexion, may mask anæmia Mucous membranes better guide, but often misleading Sallowness from constipation, acute alcoholism, etc, or natural complexion, often simulates extreme anæmia, even of mucous membranes

2 EXAMINATION OF BLOOD —Less than 5,000,000 red cells per c mm and 90 per cent hæmoglobin is anæmia

III. SECONDARY ANÆMIA

May be : (1) Acute, (2) Chronic

1. ACUTE SECONDARY ANÆMIA (post-hæmorrhagic).

Etiology.—Rapid loss of large amount of blood. Loss exceeding two pints is severe, and exceeding four pints often fatal

COMMON CAUSE.—Trauma, duodenal or gastric hæmorrhage, post partum hæmorrhage, or tubal pregnancy.

Symptoms.—(Development of symptoms watched typically in post-partum hæmorrhage) Giddiness and faintness, dyspnœa, noises

in ears, pulse becomes small and rapid. Temperature low, Patient asks for more air. Later, nausea. Convulsions rarely, with extreme loss (may be interval of some hours).

Blood Changes.—Blood shows no change immediately after hæmorrhage. Within few hours: (a) Blood is diluted by fluid drawn from tissues; (b) Red cells are discharged into the blood-stream from the marrow.

CONDITION OF BLOOD.—(1) Erythrocytes diminished (2 to 4 million); (2) Hæmoglobin reduced; (3) Colour index low, (4) Polynuclear leucocytosis; (5) A few normoblasts; some degree of poikilocytosis and anisocytosis; megaloblasts rarely.

2. CHRONIC SECONDARY ANÆMIA.

Symptoms.—Physical and mental weakness and rapid fatigue. The entire system suffers, the heart showing the earliest effects. (1) *Circulatory system*: Shortness of breath, palpitations, faintness, giddiness, and swelling of the feet. (2) *Gastro-intestinal system*: Constipation, dyspepsia, loss of appetite. (3) *Nervous system*: Headache, faintness, and giddiness, muscæ volitantes (floating specks in the vision), irritability. In women, amenorrhœa, profuse or irregular menstruation. May be slight pyrexia.

Physical Signs. (1) *Pallor*, especially of mucous membranes. (2) *Pulse*: Soft small, rapid, and easily accelerated. (3) *Heart*: Hæmic murmurs common, either at base (pulmonary area), or, less commonly, at apex. (4) Venous pulsation and murmurs in the neck common.

Blood Changes.—(1) Erythrocytes diminished (2 to 4 million). (2) Hæmoglobin reduced. (3) Colour index low, 0·4 to 0·8 i.e., hæmoglobin reduction greater than that of red cells. Poikilocytosis occurs with extreme anæmia. Normoblasts absent or scanty. Megaloblasts never present. No characteristic changes in leucocytes.

TREATMENT OF SECONDARY ANÆMIA.

When due to Extreme Loss of Blood.—First indication is to arrest hæmorrhage. Injection of physiological saline, per rectum and intravenous. Fluid by the mouth. In urgent cases, transfusion of blood (*see* PERNICIOUS ANÆMIA). Subsequent treatment as in chlorosis: rest, good diet, iron, and also arsenic.

Recovery of blood-cells and hæmoglobin often very rapid.

In Constitutional and Cachectic Conditions.—*See below*, CHLOROSIS. Iron especially increases hæmoglobin, and arsenic the number of red cells.

IV. CHLOROSIS.

A primary anæmia commencing at puberty, especially in girls, and characterized by pallor, symptoms of secondary anæmia, absence of wasting, and rapid improvement on treatment with iron.

Chlorosis, *continued.*

Etiology.—

AGE OF ONSET.—Between 14 and 20 years, during or shortly after puberty.

SEX.—Almost confined to girls; in males very rare.

DURATION.—Prolonged untreated, many years; after treatment, tendency to recurrences.

SPECIAL FACTORS.—(1) Sexual development is probably essential factor; (2) Constipation very constant and important accessory factor; (3) Poor air, overwork, and food deficient in iron.

Symptoms.—'Plumpness, puberty, and pallor.' Symptoms of secondary anæmia.

APPEARANCE.—(1) Complexion pale with a greenish tinge, whence the name chlorosis: tinge often not obvious. Sclerotics blue and eyes bright. (2) Subcutaneous fat increased.

INITIAL COMPLAINTS AND SYMPTOMS.—Easily tired. Shortness of breath. Giddiness and faintness. Palpitations. Swelling of feet. Disturbances of menstruation. Constipation. Headache. Cold feet. Emotional and nervous.

CIRCULATORY SYSTEM.—

Pulse: Rapid, easily accelerated, full and soft.

Hæmic systolic murmur common; usually maximal in second left intercostal space; less commonly at apex.

Pulsation in veins of neck, with continuous murmur over right jugular ('bruit de diable').

Circulatory symptoms: Palpitations. Cold hands and feet. Swelling of feet. Heart may be dilated.

GASTRO-INTESTINAL SYSTEM.—Appetite variable and capricious; may be desire for vinegar and acids. Flatulence and dyspepsia. Constipation almost constant. Hyperacidity of gastric contents may be present, or there may be diminution

The Blood.—

1. CHANGES IN CELLS.—(a) Red cells usually about 4,000,000 per c mm. (b) Hæmoglobin is markedly reduced (40 to 50 per cent). (c) *Colour index* very low, 0.5 or less. Thus blood is of 'secondary anæmia' type. In severe cases some poikilocytosis, anisocytosis, and a very few normoblasts, but no megaloblasts. *Main change is reduction of hæmoglobin.*

   Leucocytes may be normal in number, slightly increased, or slightly decreased, with relative lymphocytosis.

2. CHANGE IN VOLUME.—The total volume of blood is greatly increased. Hence the absolute reduction in hæmoglobin is not large, while the plasma is abnormal in amount. The more severe the anæmia the larger is the amount.

Pathogenesis.—Unknown. Probably connected with sexual development. Former theories include:—

(1) Hypoplasia of circulatory and generative organs, and small aorta (Virchow). Not supported by modern investigations.

(2) Intestinal fermentation. Bunge believed that H_2S formed

from excessive fermentation, and fixed the iron as insoluble sulphide, thus causing iron starvation. (Sulphide of iron cures as rapidly as other iron preparations.)

(3) Abnormalities of abdominal organs, e.g., gastroptosis.

Diagnosis.—The blood should always be examined to establish presence of anæmia.

Note especially. (1) Pulmonary or glandular tuberculosis. (2) Gastric ulcer. (3) Nephritis. (4) Exophthalmic goitre. (5) Neurasthenia (no anæmia).

Treatment.—Essentials are:

1. REST.—For all severe cases, in bed until blood approaches normal.
2. IRON.—A true specific remedy for the anæmia. Nature of preparation of little importance, e.g.—

℞ Pil. Ferri (Blaud's pill) gr. v.
One or two pills t.d.s. for 8 to 10 weeks.

Or,

℞ Ferri Sulphatis	gr. ij	Tinct. Zingiberis	♏x
Magnes. Sulphatis	ʒj	Inf. Gentian. Co.	ad ℥ss
Acid. Sulph. Aromat.	♏x		

t.d.s.

Reduced iron (ferri redacti, gr. ij–iv, t.d.s.), tinct. ferri perchlor. ♏x, t.d.s., and other preparations, are equally effective.

3. BOWELS must be freely opened: constipation constant. Morning saline (magnes. sulph.) effectual.
4. DIET. Moderate mixed diet. If dyspepsia, give alkalis, and commence with milk foods and light diet, then fish and meat.
5. TEETH often good, but all septic stumps to be removed.

Immediate recovery usually extremely satisfactory. Red cells reach or exceed normal, hæmoglobin recovers more slowly. Final complete recovery often necessitates several years of care and treatment.

TREATMENT DURING CONVALESCENCE AND SUBSEQUENTLY—

FRESH AIR and REGULAR EXERCISE of great importance. REGULATE BOWELS. ORDINARY MIXED DIET, including meat.

TONICS.—Arsenic, strychnine, quinine, and phosphates. Easton's syrup ℥ss to j, t.d.s. (or equivalent tablet) valuable: should be taken for long periods.

℞ Liq. Arsenicalis	♏ij	Vin. Quininæ	ad ℥ss
Vin. Ferri	ʒj		

t.d.s.

V. PERNICIOUS ANÆMIA.

(*Addisonian Anæmia*)

A fatal disease of unknown origin, characterized by intense anæmia, specific changes in the blood, and hyperplasia the bone-marrow. Considerably commoner than formerly supposed.

Pernicious Anæmia, *continued.*

Etiology.—

AGE.—Most common 35 to 45 years. Most recorded cases in extreme youth are doubtful.

SEX.—About 2 males to 1 female.

CAUSAL FACTORS.—Unknown. Most suggested factors are probably associated or secondary lesions, e.g. :—

1. ORAL SEPSIS (Hunter).—Almost invariably present, but efficient treatment does not cure disease.
2. ATROPHY OF GASTRIC MUCOUS MEMBRANE.—Achlorhydria is almost, if not quite, invariably present.

True pernicious anæmia is a disease without at present a recognizable cause. But in any series of cases or analysis of hospital records a few instances will be found in which definite factors were present which must be assumed to account for the blood changes, and in certain of these, except carcinoma, recovery can occur. The following may be mentioned :—

1. Sprue.
2. *Dibothriocephalus latus* infection. Also recorded in hookworm disease.
3. Carcinoma. Most often recorded with carcinoma of stomach, but known with other sites in the intestines. Formerly believed that secondary growths were present in bone-marrow in these cases, but this is not invariably so. Leucocytosis more common than leucopenia, and often various abnormal leucocytes.
4. Small hæmorrhages over prolonged periods, e.g., bleeding piles.
5. Pregnancy. Usually but not invariably associated with septicæmia or severe post-partum hæmorrhage. Definite and even numerous megaloblasts may be present : such cases may either die rapidly or recover completely.
6. Complete gastrectomy (Hurst).

Occasionally more or less similar lesions recorded in many circumstances—e.g., during puberty, in poisoning with T.N.T., etc. No evidence of production by syphilis or malaria.

It must be remembered : (1) Occurrence of these blood changes with above factors is *excessively rare* (except possibly with sprue and *Dibothriocephalus latus* disease) ; (2) Changes in blood are rarely quite typical of pernicious anæmia—e.g., there may be leucocytosis. Achlorhydria is almost invariable in all forms.

In some of these groups the blood has been observed to change from earlier secondary anæmia to the pernicious anæmia type, and this has also been known in apparently true pernicious anæmia.

With careful study of blood, especially in conjunction with clinical condition, errors of diagnosis are extremely rare. Any unusual feature, especially leucocytosis, must be fully considered.

Nature and Origin of Pernicious Anæmia.—Unknown. Following is working hypothesis with most support.

1) A *hæmolytic toxin* is acting slowly, in small quantities, over a prolonged period. Results : in early stages, increased output of

normal red cells as in ordinary secondary anæmia (unproved); later, as destruction continues, over-activity and multiplication of the primitive red cells (hyperplasia and metaplasia of erythroblastic tissues) in attempt to repair deficiency. The stimulus to the marrow is supposed to be 'chemotactic,' a chemical product from the destruction of red cells, and is not truly specific for red cells, as the leucoblastic tissues also react

Consistent with this theory are the following points —

(1) The condition of the bone-marrow and the excessive iron in certain sites are practically conclusive of the presence of a hæmolytic toxin

(2) Prolonged injection of small doses of ricin, a hæmolytic toxin, produces identical bone-marrow changes Large doses produce changes of secondary anæmia (Bunting).

(3) From segments of *Dibothriocephalus latus*, and from tissues in pernicious anæmia, powerful hæmolytic toxins can be extracted

The action of the toxin produces independently (1) Hæmolysis (2) Fatty degeneration · not explicable solely as the result of anæmia (3) Degeneration of the spinal cord since this may precede the anæmia On this view pernicious anæmia is not purely a disease of the blood tissues

The origin of the toxin is unknown, it has been attributed to the alimentary system, from the frequency of diarrhœa, oral sepsis, etc.

The mode of action has been variously supposed to be increase of the normal phagocytic action of the spleen and glands, or a direct alteration of the cells before they leave the marrow

CONCLUSION A *toxin* is acting over prolonged periods which causes (1) hæmolysis, (2) fatty degeneration, (3) degeneration of the spinal cord, the changes in the bone marrow being an attempt to repair the deficiencies in red cells

Morbid Anatomy.—

GENERAL Wasting slight or absent Pa[illegible] or yellow [illegible]t. Fat yellow Muscles bright red Petechial hæmorrhages [illegible]n serous surfaces May be serous effusions

HISTOLOGICAL CHANGES are essentially three (1) Fatty degeneration; (2) Excess of iron in various glands, (3) Changes in the bone-marrow

HEART. Large, soft, yellow tint, fatty degeneration extreme, especially on and near papillary muscles (yellow spots on red muscles, often referred to as 'thrush's breast')

LIVER —Normal size or somewhat large; yellow and fatty, *free iron in excess*, especially in outer zone of lobules.

SPLEEN —Always enlarged, considerable fibrosis; *free iron in excess.*

HISTOLOGY.—Cells few in number, mainly lymphocytes and phagocytes containing red cells.

KIDNEYS.—Little change.

LYMPH GLANDS.—Little change. Prevertebral ('hæmolymph') glands show great phagocytic activity.

Pernicious Anæmia—Morbid Anatomy, *continued.*

STOMACH.—Atrophy common; mucous membrane often smooth.

BONE MARROW.—Changes constant and characteristic. Typically exhibited by femur:—

MACROSCOPIC.—Red marrow throughout: no yellow fat remaining. Bone usually thin, and marrow cavity increased.

HISTOLOGY.—Great increase of megaloblasts, gigantoblasts, and primitive generations of both *red and white cells* (erythroblastic and leucoblastic): great activity of a *fœtal* type.

SPINAL CORD changes may be present (subacute combined degeneration).

Symptoms.—

GENERAL CHARACTERISTICS.—(1) *Onset insidious;* (2) Complaint of *great weakness;* (3) *Pallor marked,* often yellowish; (4) *Wasting slight or absent.*

EARLY SYMPTOMS.—(1) General weakness almost invariable; (2) Gastro-intestinal—vomiting, dyspepsia, diarrhœa; (3) Symptoms of anæmia—faintness, etc.

APPEARANCE.—*In advanced stages,* almost diagnostic: extreme anæmia with yellowish tint, combined with absence of wasting. Tint may be icteroid: occasionally brownish from use of arsenic. *In early stages,* the yellow tint is *frequently absent.*

FURTHER SYMPTOMS are divisible into two groups:—

(1) DUE TO ANÆMIA.—Languor, faintness, palpitations, breathlessness, œdema of ankles.

(2) MORE ESPECIALLY ASSOCIATED WITH PERNICIOUS ANÆMIA.—

Gastro-intestinal Disturbances:

i. Pyorrhœa alveolaris and septic teeth: almost invariable.

ii. Attacks of epigastric pain, of vomiting, or *diarrhœa.*

Nervous System.—Tingling and numbness common. Mental symptoms infrequent. (See COMPLICATIONS.)

Fever.—Rarely absent in severe stages.

Heart.—Murmurs frequent, dilatation slight.

Pulse.—Collapsing. Arteries often throbbing.

Blood-pressure.—Extremely low; often 80 to 100 mm. Hg.

Spleen.—Tip sometimes palpable.

Hæmorrhages.—(i) Large amounts (epistaxis, etc.): not frequent. (ii) Petechiæ on skin (and serous membranes). (iii) Retinal hæmorrhages: very common.

Gastric Contents.—Analysis after Ewald's test meal: (i) Free HCl absent. (ii) Total acidity extremely low (often 0 to 10 c.c. decinormal NaOH per cent). (iii) Ferments greatly reduced. That is, gastric secretion is very slight, or there is even complete achylia.

Urine.—Urobilin usually increased.

Tenderness over Long Bones.—A traditional symptom very rarely met with. Probably pains of postero-lateral sclerosis.

The Blood.—Total quantity greatly diminished *Fresh blood* appears surprisingly well coloured. Very mild *Serum* separates rapidly, and has greenish tint.

1. QUANTITATIVE CHANGES

RED CELLS Great reduction often 1,000,000 to 2,000,000 per c mm or lower

HÆMOGLOBIN – Much reduced, but not to same percentage as number of cells

COLOUR INDEX Consequently high, usually 1 or over

LEUCOCYTES Leucopenia usually 2000 to 4000 per c mm

2 CHANGES IN BLOOD-CELLS

RED CELLS —Changes constant and important, but also may occur in secondary anæmia *except the presence of megaloblasts*

(a) Poikilocytosis. pear shaped and other irregular cells

(b) Inequalities in size (anisocytosis) Extremely marked, especially large 'megalocytes', also numerous small cells

(c) Polychromatophilia cells stain blue Also basophilic degeneration blue spots

(d) *Nucleated red cells* Two types (1) Normoblasts (2) Megaloblasts

LEUCOCYTES —

(a) *Percentage of lymphocytes* high

(b) *Myelocytes* usually present, but scanty, occasionally up to 10 per cent

Turck's stimulation cells often found —viz, 'plasma cells,' cytoplasm staining dark blue, large circular central nucleus

BLOOD PLATELETS Very scanty

Summary of Blood Changes Characteristics are (1) High colour index, with great reduction in red cells and hæmoglobin, (2) Presence of megaloblasts (3) Leucopenia Except for leukæmia distinction from which is usually simple above syndrome is *proof* of pernicious anæmia Even the first factor alone is practically conclusive

Progress and Remissions.—

REMISSIONS —Great improvement or recovery' usually occurs in first attack Such 'remissions' are a distinct feature Rarely exceed three Interval before second attack often six to twelve months, subsequent remissions shorter and less complete.

BLOOD CONDITION DURING REMISSION –In first attack under observation anæmia generally improves rapidly, but rarely reaches normal e g, red cells 4,000,000, hæmoglobin 80 per cent, colour index 1 The colour index especially tends to remain high

ULTIMATE PROGNOSIS Fatal Duration one to three years, rarely longer, from first observation Acute course– a few weeks may occur, or steady descent, others remain stationary several months, then commence to fail From observations during remissions, disease has probably lasted six to twelve months before initial complaint

Pernicious Anæmia, *continued.*

Complications and Intercurrent Diseases.—SUBACUTE COMBINED DEGENERATION OF SPINAL CORD.—Frequency undoubtedly greater than formerly supposed. The symptoms may be: (i) Of postero-lateral sclerosis type, a 'spastic paralysis': increased knee-jerks, spasticity, varying degree of paralysis and sensory changes. (ii) Of tabetic type (rare). (*See* SUBACUTE COMBINED DEGENERATION OF THE SPINAL CORD.)

SEPSIS.—Frequent. Boils and local abscesses. A definite polynuclear leucocytosis may occur.

TUBERCULOSIS.—Distinctly rare.

PNEUMONIA or NEPHRITIS may occur and be fatal.

Diagnosis.—The characteristic symptoms and blood changes form generally an easy diagnosis: middle-aged patient; complains of weakness; onset insidious; extreme anæmia; wasting slight; blood shows great diminution of red cells, high colour index, leucopenia, and megaloblasts. (*See also* ETIOLOGY.)

Diagnosis from :—

1. SECONDARY ANÆMIA: By low colour index and absence of megaloblasts.
2. CARCINOMA OF STOMACH.—Rarely difficult. In gastric carcinoma: (a) Anæmia is *secondary* in type: very rare exceptions simulating pernicious anæmia are due to secondary growths in bone-marrow. (b) Wasting: onset usually rapid. (c) Presence of tumour. (d) Gastric total acidity and ferments usually not reduced so completely as in pernicious anæmia

Occasional difficulties :—

3. APLASTIC ANÆMIA, LEUKANÆMIA, and occasionally LEUKÆMIAS with high colour indices.
4. SPINAL CORD LESION.—Anæmia may be overlooked.
5. CHRONIC NEPHRITIS. MYXŒDEMA. ADDISON'S DISEASE. MYOCARDIAL HEART DISEASE.
6. DIBOTHRIOCEPHALUS LATUS (fish tapeworm) to be considered in infected districts. Blood changes identical.
7. ANKYLOSTOMIASIS.—Colour index usually low. Eosinophilia.

Treatment.—Principles are: (1) Rest in bed. (2) Diet: light mixed diet. (3) Treatment of septic teeth and mouth (avoiding indiscriminate extraction). (4) Pepsin and dilute hydrochloric acid mixtures. (5) *Arsenic.* Valuable drug. Commence liq. arsenicalis ♏ij, t.d.s., in *tumbler* of water after meals, and increase ♏j alternate days to ♏xij or ♏xv t.d.s. If signs of overdose (nausea, etc.), omit and recommence. Other preparations of arsenic may be employed—viz., sodium cacodylate, atoxyl, salvarsan: no constant superior effects.

TRANSFUSION OF BLOOD.—Some good results have followed. About 500 to 400 c.c. transferred from donor to recipient. (Four standard groups of blood are now recognized. The recipient's blood is tested against a known standard blood, and thus

'grouped' The donor must belong to the same group, or to a group which is known to be harmless to the recipient's group)

SPLENECTOMY —Based upon (1) Good results in splenic anæmia Banti's disease, and congenital family jaundice; (2) Destruction of red cells in spleen Shows no obvious good results

A vast number of drugs and treatments have been employed It must be recollected that the majority of cases have a distinct 'remission or apparent recovery' without specific treatment

VI. APLASTIC ANÆMIA.

A fatal disease closely resembling pernicious anæmia clinically but distinguished from it by absence of nucleated and abnormal red cells in the blood, and by atrophy or aplasia of bone-marrow A rare disease

Principal Characteristics (for comparison with pernicious anæmia) —

1 ETIOLOGY —*Age* young adults rare after 40 *sexes* equal Occurred in trinitrotoluene poisoning

2 MORBID ANATOMY *Bone marrow* Yellow marrow alone present *little or no red marrow left* may be phagocytes containing erythroblasts erythrocytes and debris (evidence of destruction of blood) In liver and spleen, *free iron usually absent*, and when present amount small

3 SYMPTOMS identical with pernicious anæmia but more rapid and *no remissions* Tendency to purpura and large haemorrhages

4 BLOOD CHANGES Correspond to aplasia of bone-marrow

a. LEUCOCYTES Extreme leucopenia (500 to 2000), with relative lymphocytosis

b NUMBER OF RED CELLS Very low 500,000 to 1,500,000 per c mm

c APPEARANCE OF RED CELLS Normal *no nucleated* RBCs, *poikilocytosis, etc., present*

d COLOUR INDEX Variable above unity or low

e BLOOD PLATELETS Very scanty

f SERUM - Colourless

5 DURATION Rarely exceeds 6 and may not exceed 3 months

Pathogenesis.—The absence of changes in the red cells and absence and small amount of free iron in the liver and spleen suggests that the action is not that of a hemolytic toxin but directly destructive on the marrow

Trinitrotoluene (T.N.T.) Poisoning.*

Symptoms —Cyanosis is common among workers, even in absence of anæmia nitroxy and methæmoglobin in blood Fatal cases are due to —

* Pathological studies, especially by P N Panton, Matt A Stewart, and H. M Turnbull, Royal Society of Medicine 1917, and Government publications

Trinitrotoluene Poisoning, *continued*

1 TOXIC JAUNDICE — Jaundice Hæmorrhages common Aplastic anæmia never present at onset Milder forms of jaundice recover *Morbid Anatomy*: Acute yellow atrophy of liver

2 APLASTIC ANÆMIA Rarer than above Not necessarily preceded or accompanied by toxic or simple jaundice

A long interval may occur between exposure to T N T and onset of toxic jaundice, and even longer (many months) in case of aplastic anæmia

CHAPTER XCIX.

LEUCOCYTOSIS AND LEUCOPENIA.

The variations in the number and percentage of those white cells which are *normally* present in blood, and their principal causes are as follows: —

Polynuclear Leucocytosis. Increase both in (1) total leucocytes and (2) percentage of polynuclear cells The increased percentage has a diagnostic importance equal to or greater than that of the total numbers Occurs in

(1) Acute inflammations infections and specific fevers
Exceptions Tuberculosis typhoid malaria measles, mumps, and influenza *in absence of complications*

(2) Severe or generalized stages of many cachectic conditions e g, glandular tuberculosis carcinoma of stomach and intestines

Following splenectomy, for a variable period

RELATION TO CLINICAL CONDITION —

1 OF MODERATE SEVERITY — Degree both of total increase and percentage varies with severity of infection

2 OF EXTREME SEVERITY (e g, virulent septicæmia) — No increase, or even marked decrease in total leucocytes *percentage* of polynuclear cells usually very high (85 to 95)

Lymphocytosis. —Increase both in (1) total leucocytes, and (2) percentage of lymphocytes almost invariably small lymphocytes Occurs in (1) Leukæmia, (2) Whooping cough, (3) An obscure group of infections e g, glandular fever In infants percentage of lymphocytes is normally high (about 40) up to age of six years and frequently increases in pyrexial conditions

Leucopenia.—Diminution in total leucocytes, usually *with relative lymphocytosis* Occurs in (1) Certain specific infections, especially protozoal (2) Certain blood conditions splenic anæmia pernicious anæmia, aplastic anæmia (3) Not uncommon in chronic glandular lesions tuberculosis lymphadenoma

.—Over 4 per cent is an excess; may rise to 50 per cent or higher. Occurs in following, but not invariably :—

1. *Intestinal parasites*: with common parasites, including hydatid cysts; marked with ankylostoma, trichinella, filaria, bilharzia.
2. *Skin diseases*, e g., psoriasis.
3. *Bronchial asthma* (for a period after attacks).
4. *Leukæmia.*
5. *Lymphadenoma*: slight and occasional, and insufficient for diagnosis.

Increase of Mast Cells.—Occurs in myeloid leukæmia (usually atypical mast cells).

(For notes on leucocytes, *see* LEUKÆMIA.)

CHAPTER C.

THE LEUKÆMIAS.*

Leukæmia is a disease of the blood-forming tissues. There are two main hæmopoietic systems: (1) Bone-marrow or myeloid tissue: concerned in formation of granular leucocytes and red cells. (2) Lymphoid tissue, comprising spleen, lymphatic glands, and possibly all small accumulations of lymphoid tissue: concerned in formation of non-granular leucocytes or 'lymphocytes.' Leukæmias can be classified on this basis: –

1. Myeloid leukæmia: (a) Acute (myeloblastic), (b) Chronic.
2. Lymphoid leukæmia: (a) Acute; (b) Chronic.

The entire hæmopoietic system is affected in all types. Of pathogenesis, nothing is known.

Clinically, little difference exists between the acute forms of the two types in symptoms, physical signs, and prognosis. So also for the chronic forms. Thus a valuable clinical differentiation is into

1. Acute leukæmia: (*a*) Myeloid; (*b*) Lymphoid.
2. Chronic leukæmia: (*a*) Myeloid; (*b*) Lymphoid.

'Mixed' Leukæmias.—The existence is doubtful of a leukæmia partly myeloid and partly lymphoid. Recorded cases usually fail to distinguish lymphocytes and myeloblasts.

Alteration from Myeloid to Lymphoid Form.—Not authenticated.

Notes on Leucocytes with reference to Leukæmia.—The predominant cell may be of various types, having its origin either in lymphoid or bone-marrow tissue.

PAPPENHEIM'S VIEWS ON DEVELOPMENT OF BLOOD-CELLS.—Pappenheim calls the myeloblast 'lymphoidocyte', and considers that it is the precursor alike of polynuclear cells, lymphocytes, red cells, and large mononuclears, i.e., is the common primitive blood-cell, developing from the endothelial cells lining

* *Quarterly Journal of Medicine*, July, 1914.

The Leukæmias—Notes on Leucocytes, *continued.*

the walls of capillaries. The subsequent cycle of development of the lymphoidocyte depends on various stimuli. In general the stimulus arriving at such hæmopoietic tissue in bone-marrow results in the ultimate production of polymorphonuclear cells, while the stimulus in lymphoid tissue results in lymphocytes; but this is not invariably so, and the bone-marrow may (if the stimulus demands it) produce lymphocytes—e.g., in lymphoid leukæmia—or the lymphatic glands may produce polynuclear cells—e.g., in very acute infections.

Pappenheim's theories uphold the unity of all hæmopoietic tissues.

LYMPHOIDOCYTES OR MYELOBLASTS.—Non-granular mononuclear cells; large nucleus, with definite chromatin structure, containing several distinct *nucleoli*, and surrounded by zone of deep-blue cytoplasm. Not present in normal blood, but numerous in bone-marrow. Two types occur:—

LARGE MYELOBLASTS.—Broad zone of cytoplasm; nuclei stain comparatively lightly, and nucleoli are easily recognized.

SMALL MYELOBLASTS.—Resemblance to small lymphocytes often close.

Occurrence of Myeloblasts in Blood.—(1) Myeloblastic leukæmia. (2) Chronic myeloid leukæmia: invariably present, usually in small numbers (a few per cent). (3) Lymphoid leukæmia: in very small numbers. (4) Occasionally, in very small numbers, in conditions of great activity of bone-marrow, e.g., severe leucocytosis.

MYELOCYTES.—Granular mononuclear cells of bone-marrow origin. Are precursors of polynuclear leucocytes, and intermediate and transitional forms occur. *Granules* may be fine neutrophil, eosinophil, or basophil (and rarely amphophilic, all types being present). *The nucleus* is oval or circular; in intermediate forms, horseshoe or showing signs of division. No nucleoli are present. The common type is the finely granular *neutrophil myelocyte.*

Occurrence of Myelocytes in Blood.—(1) Chronic myeloid leukæmia: is characteristic cell. (2) In small numbers in all conditions of great activity of bone-marrow—viz., myeloblastic leukæmia, severe leucocytosis (sepsis), pernicious anæmia, etc.

TRANSITIONAL STAGES.—Cells occur which are intermediate stages between myeloblasts and myelocytes, and also between myelocytes and normal leucocytes. In general their numbers are small, but occasionally in leukæmia the predominant cell may be of such type—e.g., a late myeloblast, possessing some granules, with absence of nucleoli or similar feature; or a cell intermediate between the myeloblast and large hyaline (large mononuclear) of normal blood, a type known as the Rieder cell.

LYMPHOCYTES.—In normal blood two forms occur, 'small' and 'large'. These are distinct types, and the difference is not merely one of size. Transitional stages between lymphoidocytes and lymphocytes are difficult to trace.

SMALL LYMPHOCYTE.—Nucleus stains very dark; contains irregular masses of chromatin, and is surrounded by a narrow ring of dark-blue cytoplasm. Almost invariable type in acute lymphoid leukæmia, and usually in chronic form. Sometimes cell in leukæmias is rather larger than normal, with an indented nucleus.

LARGE LYMPHOCYTE.—Nucleus stains lighter, with definite chromatin network, and is surrounded by a broad ring of light-blue cytoplasm; scanty 'azur' granules usually present. Occurrence extremely rare (if ever) in acute leukæmia, rarely in chronic lymphoid leukæmia, forming the most chronic type.

Summary of Leukæmias in Relation to Type of Cell.—

1. MYELOBLASTIC.—(i) Small cell: acute. (ii) Large cell: very acute.
2. MYELOID.—(i) Acute: (*a*) Acute myelocytic very rare. (*b*) Myeloblastic (*see above*). (ii) Chronic: commonest leukæmia: may terminate as myeloblastic.
3. LYMPHOID. (i) Small cell: acute or chronic, usually subacute. (ii) Large cell: very chronic, very rare: extremely rarely acute.
4. ABNORMAL LEUCOCYTES.—Acute, e.g., hyaline or Rieder cell type. Cells are akin to myeloblasts.

Relative Frequency of Different Types.—Chronic myeloid leukæmia is as common as all other types together. The relative frequency of the two acute types is at present uncertain, owing to frequent confusion. The chronic lymphoid type is very rare. The different varieties are discussed below under the following headings:—

1. CHRONIC MYELOID LEUKÆMIA. Described fully.
2. CHRONIC LYMPHOID LEUKÆMIA.
3. ACUTE LEUKÆMIA. The common symptoms, signs, etc. of the two types are described, followed by separate descriptions of the blood changes.
4. VARIOUS ATYPICAL CONDITIONS RESEMBLING LEUKÆMIA.—Leukanæmia, pseudoleukæmia, chloroma.

VI. CHRONIC MYELOID LEUKÆMIA.

(*Splenomedullary Leukæmia.*)

A fatal disease affecting the bone-marrow, and characterized by great increase and abnormality of the bone-marrow cells in the blood and enlargement of the spleen.

Etiology.—

SEX.—About 2 males to 1 female.

AGE.—Any decade, commonly 25 to 40 years, rare under 20 years. No predisposing factors known. Never produced experimentally

Chronic Myeloid Leukæmia, *continued.*

Morbid Anatomy.—Important lesions are confined to the hæmopoietic system.

BONE-MARROW.—Medullary cavity occupied by grayish-red tissue; no fat remaining.

HISTOLOGY.—Great hyperplasia of leucoblastic (leucocyte-forming) tissues. Note:—

1. Numerous non-granular large mononuclear cells, viz., myeloblasts.
2. Numerous myelocytes, frequently showing mitosis.
3. Nucleated red cells, both normoblasts and megaloblasts.

SPLEEN.—Always enlarged, often enormous: commonly about 10 pounds.

SURFACE.—Perisplenitis and adhesions common; capsule thickened; veins in hilus enlarged.

ON SECTION.—Tough from sclerosis; general red surface, often scattered gray areas from old infarcts.

HISTOLOGY.—Resembles bone-marrow; no Malpighian corpuscles remain; enormous numbers of leucocytes with numerous myelocytes present. Changes may be transformation into leucoblastic tissue, or the result of infiltration with cells, probably the former.

LYMPHATIC GLANDS.—Peripheral glands usually unaffected. Mesenteric glands often enlarged. Changes resemble spleen. Occasionally enlarged glands have green tint on section. Solitary follicles, Peyer's patches, etc., may be swollen by leucocytes.

BLOOD.—Grayish-colour from excess of leucocytes, often clotted.

OTHER TISSUES.—

LIVER.—Enlarged; widespread leucocytic infiltration, distending capillaries; in microscopic sections, resembles miliary abscesses.

KIDNEYS AND LUNGS.—Similar leucocytic infiltration.

HEART.—Blood-clots very common: appearance may resemble pus.

Symptoms.—Onset is insidious.

1. ENLARGEMENT OF ABDOMEN (BY SPLEEN).—Usually earliest symptom. Occasionally: weakness or breathlessness.
2. ANÆMIA.—*Not marked* in early stages, but increases later.
3. WASTING.—May be rapid.

OTHER SYMPTOMS.—

RETINAL HÆMORRHAGES.—Almost constant. 'Leukæmic retinitis': fundus pale, hæmorrhages, white spots. Sight may or may not be affected. In rare cases is *earliest symptom.*

FEVER.—Usually slight, irregular, or transient pyrexia.

COUGH.—Rarely troublesome. In later stages from effusion (usually on left) or œdema at bases. Breathlessness in earliest stages from pressure of spleen or anæmia.

ŒDEMA OF LEGS.—Common. Rarely ascites.

URINE.—Enormous excretion of uric acid, from destruction of leucocytes. No gout or 'uric-acid symptoms'.

MÉNIERE'S DISEASE.—Sudden onset, from hæmorrhage into semicircular canals.*

Very rare: Skin tumours. Priapism (traditional symptom).

ENLARGEMENT OF SPLEEN.—Invariable; usually reaches umbilicus or beyond; edge and notch easily felt; surface smooth, may be tender. Varies in size, roughly with number of leucocytes

LIVER.—Generally palpable

LYMPHATIC GLANDS.—Not usually enlarged. Most commonly axillary.

TERMINAL STAGE. Certain symptoms are *infrequent* during chronic stages, but often important *towards termination*, especially if this is acute:

HÆMORRHAGES.—Especially nose or gums. Rarely severe until late stages.

PURPURA.—Almost confined to acute termination, *very rare* in chronic stage.

GASTRO-INTESTINAL DISTURBANCES. Vomiting, diarrhœa, etc. Not uncommon towards end.

The Blood.—Changes are characteristic and pathognomonic. Fresh blood, in severe cases, is grayish red from excess of leucocytes

LEUCOCYTES. *Total number greatly increased*: commonly 200,000 to 300,000; may exceed 1,000,000. Total number of all varieties is increased

CHARACTERISTICS (in stained blood)

1. Presence of abnormal granular bone marrow cells in large numbers, i.e., *myelocytes*. Percentage 5 to 25, less commonly up to 40 or 50
2. Increase of mast cells, and usually of eosinophils

Transitional forms between myelocytes and normal leucocytes are numerous. In some cases very few cells are truly normal, the nucleus showing but slight division

Mast cells (coarsely and finely granular basophils) usually in large numbers, forming 5 to 10 or even 25 per cent.

Eosinophils.—Percentage increased.

Myeloblasts. Invariably present, usually not exceeding 10 per cent.

ERYTHROCYTES. Number in early stages not greatly diminished; may be normal. Falls as condition advances.

Colour index usually low, 0.6 to 0.8

Nucleated red cells, normoblasts and megaloblasts, rarely absent, may occur even in absence of severe anæmia.

Blood-platelets very numerous.

SUMMARY OF CHARACTERISTIC CHANGES—

1. Total number of leucocytes greatly increased.
2. Increase mainly due to granular cells.
3. Presence of myelocytes and primitive bone-marrow cells.
4. Presence of normoblasts and megaloblasts.

* Occurred in Ménière's original case.

Chronic Myeloid Leukæmia, *continued.*

Course and Prognosis.—

INTERCURRENT DISEASES.—Rare. Death generally directly from the disease. With sepsis, leucocytes may temporarily diminish in number. Tuberculosis, pneumonia: rare.

COURSE AND DURATION.—*Recovery never occurs.* Duration *before* observation, probably about 1 year; under observation, usually 1 year; rarely exceeds 3 years. Two groups can be recognized:—

1. UNDER 35 YEARS.—Tendency to great variations in numbers of leucocytes and general condition. Response to X rays usually marked, general improvement temporarily occurring. But this group has shorter duration and acuter course. 'Aleukæmic intervals' may occur, when blood is practically normal, but *mast cells always remain abnormal.* Myeloblastic termination may occur.
2. OVER 35 YEARS.—Under treatment, little change in blood and general condition, but duration is longer than in previous group.

MYELOBLASTIC TERMINATION.—Occasionally high percentage of myeloblasts appears suddenly (40 to 98 per cent); usually total number of leucocytes low, often marked leucopenia (1500 to 4000); but may be 20,000 to 100,000. Death always *within few days*, with terminal symptoms as above, i.e., is proof of greatly exhausted bone-marrow. Rarely observed, owing to short duration, and frequency unknown.

Diagnosis.—Usually simple. Enlargement of the spleen results in examination of the blood.

Treatment.—

GENERAL TREATMENT.—Good food, etc.

SPECIFIC TREATMENT, designed to reduce spleen and number of leucocytes.—Of most efficiency are: (1) X rays, (2) Arsenic, (3) Benzol.

X RAYS.—Applied to spleen and long bones. Often cause great reduction in leucocytes and spleen, and improved general condition, but response to treatment differs essentially in the two groups separated under PROGNOSIS.

ARSENIC.—Similar but slighter action.

BENZOL.—In capsules with olive oil. Commence with 10 minims twice a day; increase to 50 to 75 minims daily. Results hopeful. Causes reduction of leucocytes in certain cases.

DURING SPECIFIC TREATMENT: *Examine blood regularly*; always discontinue if number falls to 10,000 or myeloblasts increase considerably.

SURGICAL TREATMENT.—Removal of spleen. Rapidly fatal if spleen large; and when recovery from operation has occurred, subsequent course of disease apparently unaffected.

II. CHRONIC LYMPHOID LEUKÆMIA.

Clinically indistinguishable from chronic myeloid leukæmia, but enlargement of lymphatic glands is rarely absent. Very rare.

Etiology.—

AGE.—Usually later decades, 40 to 70.
SEX.—About 2 males to 1 female.
No predisposing factors known.

Morbid Anatomy.—The general condition of the organs corresponds to chronic myeloid leukæmia, but the tissues, including bone-marrow and spleen, are packed with lymphocytes.

Symptoms (*see* CHRONIC MYELOID LEUKÆMIA).—*Enlargement of abdomen by spleen* may attract attention. Occasionally *weakness* and *wasting* noticed first. Enlargement of lymphatic glands usually prominent. All other symptoms rare
Anæmia not marked at onset: may become extreme
Skin lesions occur, rarely, but more often than in other leukæmias.

Blood.—

LEUCOCYTES.—(1) Total count: 60,000 to 100,000 per c mm., may be very high. (2) Lymphocytes usually over 90 per cent, generally of small type; the very rare form with large lymphocytes is the most chronic of all leukæmias A few myeloblasts may be present.
ERYTHROCYTES. Progressive anæmia as in chronic myeloid leukæmia.

Course and Prognosis.—Most chronic leukæmia. May be 2 to 10 years Death from weakness and anæmia No acute myeloblastic termination has been observed *

Treatment.—In correspondence with the more chronic group of myeloid leukæmia occurring after age of 35 years, these cases usually show little or no response to X rays or arsenic.

III. ACUTE LEUKÆMIA.

An acute fatal disease characterized by the presence in the blood of a high proportion of mononuclear or closely similar cells.

Various types of cells are present in different cases, constituting: (1) Acute myeloblastic or myeloid leukæmia, (2) Acute lymphoid leukæmia; (3) Other rarer acute leukæmias, e.g., chloroma.

All types of acute leukæmia agree in: (1) The clinical symptoms, signs, and progress; (2) The changes in the red cells; (3) The fact that the leucocytes include a high percentage of some mononuclear or closely similar cell. The diagnosis of acute leukæmia is usually made readily, from examination of a stained blood-film. The determination

* The recognition of a change from small lymphocytes to small myeloblasts would probably be difficult, but no evidence of such a change has been obtained. This difficulty would not apply to large myeloblasts, nor to the group with large lymphocytes; but this latter is very rare.

Acute Leukæmia, *continued.*

of the type of the predominant cell is of pathological interest, but not of clinical importance. All forms are rare. The various types are discussed after the general clinical description.

Etiology.—

AGE.—Usually under 20 years.
SEX.—About 2 males to 1 female.
No predisposing diseases or factors.

Symptoms and Signs.—Prominent features, any one of which may first attract attention, are:—

1. PALLOR.—Anæmia is always severe even at first examination, and becomes extreme.
2. SWELLING AND ULCERATION OF GUMS, also cheek, tonsils, etc.—Often great severity.
3. HÆMORRHAGE.—Frequency: gums, nose, stomach, rectum; in females often vaginal.
4. PURPURA.
5. ENLARGEMENT OF LYMPHATIC GLANDS.—Occurs in most cases, but is rarely very great.

Other features are:—

6. ENLARGEMENT OF SPLEEN. Palpable in 75 per cent, but never attracts attention initially; usually slight, rarely reaches to umbilicus finally. LIVER usually enlarged
7. VOMITING.—Often intractable towards end. Diarrhœa less common.
8. FEVER.—Rarely absent: often 103° to 104°.

Course.—Initial symptom of pallor, with or without oral symptoms and hæmorrhage, may be followed rapidly by enlargement of spleen and glands and by hæmorrhages and purpura. In some cases glands and spleen do not enlarge throughout. Disease progresses continuously, and weakness increases rapidly, especially in this latter group. Remissions are rare. Vomiting usually troublesome. General condition of extreme discomfort.

Duration.—Death occurs in few days to few weeks from date of observation. Total duration of illness: about 3 weeks to 3 months.

Prognosis.—Death invariable in short period. Sole factor in prognosis is the exclusion of chronic lymphoid leukæmia.

Diagnosis.—Symptoms usually, but not invariably, lead to examination of the blood; recognition simple from great predominance of a mononuclear cell.

Condition clinically may be confused with:—

1. PURPURA, SCURVY.—*Note*: Purpura in absence of a palpable spleen is practically never of leukæmic origin.
2. ANGINA.—In any case of ulceration within mouth or swelling of gums *which is resistant to treatment*, the blood should be examined.

3. INFECTIVE ENDOCARDITIS, SEPTICÆMIA, ETC. From presence of purpura and pyrexia.
4. HÆMORRHAGIC FORMS OF ACUTE SPECIFIC FEVERS.
5. TYPHOID OR TYPHUS.—Owing to toxic condition, high temperature, and palpable spleen.

Lymphoid leukæmia may be suggested by certain conditions producing lymphocytosis:—

WHOOPING-COUGH.—Lymphocytes may form 80 to 90 per cent of leucocytes (transient).

INFLAMMATION OF LYMPHATIC GLANDS.—With adenitis of any origin (e.g., tuberculous, glandular fever), especially in children, there may be considerable increase of lymphocytes.

Treatment.—*Palliative.* All symptoms very resistant. For the mouth, hydrogen peroxide wash. X rays, arsenic, etc., valueless; latter usually causes or increases vomiting.

VARIETIES OF ACUTE LEUKÆMIA.

Distinguished by the leucocytes: (1) Myeloblastic leukæmia: (*a*) Primary; (*b*) Secondary, acute termination of chronic myeloid leukæmia. (2) Acute lymphoid leukæmia. (3) Atypical leukæmias

1. Myeloblastic Leukæmia.

THE BLOOD. Changes similar in primary and secondary forms.

a LEUCOCYTES.

i. Number: 30,000 to 200,000 per c.mm. or higher. May be leucopenia, especially in secondary group.

ii. Cells: *Predominant cell is a myeloblast*, large or small type; may form 90 per cent or more of total cells

b ERYTHROCYTES. Extreme anæmia, both cells and hæmoglobin: advances rapidly. Colour index often high. Normoblasts and megaloblasts: numbers vary, occasionally very numerous.

MORBID ANATOMY. Spleen and lymphatic glands usually enlarged; there may be a *green* tint on opening glands, evanescent. Bone-marrow red or grayish.

Lipæmia may be present during last few days; serum is opaque milky colour; occasional cause of milky blood. (More correctly, is a *pseudo-lipæmia*, the body present being not fat, but a proteo-lipoid, soluble in alcohol but not in ether.)

ACUTE MYELOCYTIC LEUKÆMIA.—Acute course, but blood as in chronic myeloid leukæmia throughout—viz., myeloblasts not exceeding 2 to 10 per cent. Very rare.

CONFUSION OF LYMPHOID AND MYELOBLASTIC LEUKÆMIAS. *Myeloblasts* have frequently been mistaken for lymphocytes. Large myeloblasts, (especially atypical varieties) form the most acute leukæmias, accounting for former statements that with large lymphocytes leukæmia is acute, and with small it is chronic (*see* SUMMARY OF LEUKÆMIAS, p. 61[illegible]).

Varieties of Acute Leukæmia, *continued.*

2. **Acute Lymphoid Leukæmia.**—

THE BLOOD.—

a. LEUCOCYTES.—

i. Number: Usually *under* 10,000 at first observation; leucopenia common, 2000 to 5000. Less commonly, 20,000 to 100,000.

ii. Cells: *Predominant cell is a lymphocyte,* almost invariably of small type; may exceed 99 per cent of total. A few myelocytes usually present, and (on careful search) stray myeloblasts.

b. ERYTHROCYTES.—*Severe anæmia from onset,* both cells and hæmoglobin; advances rapidly, may fall under 1,000,000 and 20 per cent hæmoglobin. Colour index may be high. Normoblasts rarely absent, but numbers generally small; occasionally megaloblasts.

Rapid variations in leucocytes may occur, even to within normal limits; anæmia and general condition not showing corresponding improvement.

MORBID ANATOMY.—Spleen and lymphatic glands enlarged. Bone-marrow increased, little fat, colour red. Hæmorrhages on serous membranes.

HISTOLOGY.—All hæmopoietic tissues contain large numbers of the cell predominating in the blood. Liver may give free iron reaction.

Atypical Leukæmias.—Rare. Characterized by presence of a *predominant* cell differing from the usual types of myeloblast, myelocyte, and cells of normal blood. The majority are varieties of myeloblasts, and the condition is usually very acute.

ACUTE HYALINE LEUKÆMIA.—Cells resemble, often closely, the large hyaline of normal blood ('large mononuclear'). Clinically: very acute, general glandular enlargement, severe hæmorrhages, but absence of purpura. Types occur with cells intermediate between myeloblasts and large hyalines (Rieder cells).

CHLOROMA.—A form of myeloblastic leukæmia with special manifestations. (*See* p. 626.)

IV. ATYPICAL CONDITIONS RESEMBLING LEUKÆMIA.

Many conditions occur in which changes in the hæmopoietic tissues and alterations in the blood are suggestive of, though varying in some degree from, leukæmia. Many grades occur, and each grade has received many different names. Practically, very little is known of these groups. The following conditions may be noted:—

1. Growths in or of Hæmopoietic Tissues which are Neoplastic or Suggestive of Neoplasms.—Any growth in these tissues may alter the condition of the blood; thus, in glandular tuberculosis or lymphadenoma there is some leucopenia and relative lymphocytosis. Apart from these are:—

a. AFFECTIONS OF LYMPHOID TISSUE.—(i) True sarcoma, and various stages of less definite and ill-defined sarcomatous growths—e.g., 'lymphosarcoma,' 'lymphoma': some blood change is common—e.g., relative lymphocytosis, with leucopenia or sometimes leucocytosis (causing greater difficulty). (ii) At other end of scale is *chloroma* (*see* p. 626): blood typical of leukæmia, but glandular growths definitely *infiltrate other structures*—i.e., possess malignant characteristics. (iii) Between these groups are forms resembling lymphoid leukæmia, with lymphatic tumours showing varying tendencies to infiltration (Sternberg's leucosarcoma).

b. AFFECTIONS OF MYELOID TISSUE.—Neoplasms and secondary growths in marrow may cause: (i) Anæmia of secondary or primary type; (ii) Leucocytic changes, with presence of myelocytes and immature bone-marrow cells. Thus, pernicious anæmia may be simulated—e.g., in gastric carcinoma (rarely); or condition may somewhat suggest myeloid leukæmia.

'*Myeloma*'. Growths in bone-marrow, associated with Bence-Jones albumosuria,' but no blood changes.

2. '**Leukanæmia**' (Leube). *Conditions combining pernicious anæmia and leukæmia* viz.: (*a*) Leucocytic changes suggesting leukæmia (lymphoid or myeloid), (*b*) Erythrocytic changes suggesting pernicious anæmia.

CLINICALLY.—Rapidly fatal course, with symptoms as described in 'acute leukæmia' (ulceration of mouth, hæmorrhages, purpura, etc.).

NATURE OF CONDITION.—Note:—

a. In acute leukæmia, the erythroblastic tissues also suffer, and rapid anæmia occurs, with normoblasts, megaloblasts, and often high colour index. This accounts for most recorded cases of 'leukanæmia'. Even in chronic myeloid leukæmia, megaloblasts are almost constant.

b. In pernicious anæmia, there is histological evidence of over-activity of leucoblastic as well as erythroblastic tissues, and myelocytes are usually present in the blood. In view of histological changes in the marrow, the low number of myelocytes is surprising.

c. In recorded cases (e.g., Leube's own), the number of leucocytes is usually low, as occurs also in some 'acute leukæmias'.

CONCLUSION.—Recorded cases of 'leukanæmia' can be classified under various other blood conditions.

3. '**Pseudo-leukæmia.**'—*Tissue changes of leukæmia, with blood normal or little changed.* Includes a series of various types. The tissue changes may resemble, slightly or closely, myeloid or lymphoid leukæmia; total leucocytes in blood usually normal, but with some lymphocytosis or myelocytes, or in other cases normal.

Atypical Conditions resembling Leukæmia, *continued*

The group shows relations to, and cases may be difficult to distinguish from lymphadenoma, tuberculosis of glands, and remissions in myeloid leukæmia and pernicious anæmia

CHLOROMA.

A form of 'acute leukæmia' characterized by (1) Infiltration of subperiosteum and other tissues by the marrow cells (2) Predominance in skull bones, and (3) Green colour of growth on section

Morbid Anatomy.—

SITES especially affected are (1) *Orbit*, (2) *Temporal bones* (3) Vertebræ, (4) Kidneys Other bones, *especially skull*, also liable Also *lymphatic glands* and skin

TUMOURS are formed of masses of cells resembling those of acute myeloblastic leukæmia

GREEN TINT may be very bright fades on exposure to air nature unknown Not invariably present Similar slighter tint may occur in acute myeloblastic leukæmia

Symptoms – Those of acute leukæmia with certain local and pressure signs –

1 OF ACUTE LEUKÆMIA Severe rapid anæmia wasting purpura hæmorrhages swelling of gums vomiting etc *Spleen and lymphatic glands usually enlarged* and may be greatly so

2 PRESSURE SYMPTOMS Characteristic are (a) *Protrusion of eyeballs* (from growth in orbit) (b) *Swellings in temporal region* (c) *Blindness* Often deafness

3 TUMOURS FROM ENLARGED GLANDS

Blood Changes As in acute myeloblastic leukæmia

Prognosis and Duration –Always fatal Duration 3 to 6 months

CHAPTER CI

HODGKIN'S DISEASE.

(*Lymphadenoma*)

A fatal disease characterized by enlargement of the lymphatic glands and progressive anæmia, usually with enlargement of the spleen

Etiology.—

AGE —Commonest in young adults

SEX. —Proportion of 2 males to 1 female

Pathogenesis. —Uncertain Generally considered to be a *granuloma*, the result of a chronic inflammation Various bacteria have been described, none confirmed, transmission to animals fails (experiments not very satisfactory)

RELATION TO TUBERCULOSIS.—Lymphadenomatous glands may become tuberculous, but in early stages tubercle bacilli are not present, and the histological changes are specific.

Morbid Anatomy.—The characteristic change results in enlargement of the lymphatic glands and lymphoid tissue. May be enormous masses, but the nodules are discrete, united by connective tissue, rupture of the capsule being rare. *Periadenitis* may follow secondary infection or as result of X rays.

1. LYMPHATIC GLANDS.—
   DISTRIBUTION OF AFFECTION AND ENLARGEMENT.—(a) *Superficial glands* commonly affected first - most often cervical; at onset may be unilateral. (b) Glands of axilla and groins usually enlarge next. (c) Internal glands; when these are affected together with the superficial glands, enlargement is usually in order from above downward, e.g., mediastinal before abdominal; in some cases the internal glands alone are affected. (d) Lymphoid tissue in all sites may be affected, e.g., in intestinal canal.
   ON SECTION. Gray surface, translucent appearance, often lobulated by strands of connective tissue; there may be yellow areas of fatty degeneration, but typically without caseation.
   HISTOLOGY. Changes pathognomonic, and form only definite method of diagnosis: (a) *Giant cells* with 3 or 4 nuclei ('lymphadenoma cells'); (b) Great increase of endothelial cells having large single nuclei; (c) *Eosinophils* in large numbers and masses.
2. SPLEEN.—Always enlarged to some extent, but never extreme.
   ON SECTION.—Contains gray areas about the size of a walnut, histologically resembling the structure of the abnormal lymphatic glands ('hard-bake spleen').
3. LIVER. Often enlarged; may be nodules like those in spleen.
4. KIDNEYS.—Occasionally contain nodules.
5. BONE-MARROW.—Infiltrated with similar lymphoid tissue.

All lymphoid tissue may be similarly affected.

Blood Changes.—

1. ERYTHROCYTES. Progressive secondary anæmia: reduction in red cells and hæmoglobin, and lowered colour index. Changes often slight in early stages, but finally severe.
2. LEUCOCYTES.—There may be either: (a) Leucopenia, with a relative lymphocytosis: commonly. (b) Leucocytosis, with an increase of polynuclear cells: especially in terminal stages. *Eosinophils* may be increased, rarely exceeding 10 per cent.

   Similar changes (except eosinophilia) occur in glandular tuberculosis and certain other causes of general lymphatic enlargement, and are not diagnostic.

Symptoms.—

ONSET.—Insidious.

Hodgkin's Disease—**Symptoms,** *continued.*

EARLIEST SYMPTOM—Usually enlargement of glands, especially cervical, painless, size attracts attention

PALLOR, ANÆMIA, and WEAKNESS—Slight in early stages, slowly increase

SPLEEN—Usually palpable (75 per cent), a few finger breadths edge hard and sharp

FEVER—Usual, slight and irregular (*see also* PEL-EBSTEIN SYNDROME, p 629)

BOILS PRURITUS not uncommon BRONZING OF SKIN occasionally (intra-abdominal glands)

ENLARGEMENT OF GLANDS -

CHARACTER—*Glands discrete*, freely movable (until mass very large), *never adherent to, and no reddening of, skin* no ulceration or caseation Glands soft when growth rapid firm and hard when slow

Note - Periadenitis from secondary infection or X rays may cause glands to adhere to each other, but not to skin, X rays may redden skin

DISTRIBUTION (*see* MORBID ANATOMY) Commonly commences in posterior triangle of neck Finally may affect all lymphoid tissue and produce enormous masses

Scattered lymphoid tissue which may be affected includes (1) Glands over sternum and below clavicles common Also epitrochlear gland (2) Gastro intestinal canal producing diarrhœa and other disturbances (3) Lungs cough, areas of consolidation (4) Spinal canal producing pressure on spinal cord (5) Intracranial symptoms of tumour rare

PRESSURE SYMPTOMS Numerous pressure effects may occur —

1. CERVICAL GLANDS - Pressure on trachea causing cough and dyspnœa, increasing, and finally fatal Various other effects Dysphagia, inequality of pupils, protrusion of eyes (sympathetic nerve), œdema of face, paralysis of recurrent laryngeal nerves
2. AXILLARY GLANDS—Pain and œdema of arms
3. MEDIASTINAL GLANDS—Signs of intrathoracic tumour especially cough, dyspnœa, and cyanosis, occasionally œdema and dilated veins Rarely pleural effusion (may be chylous)
4. RETROPERITONEAL GLANDS—Abdominal pain, may simulate appendicitis, tuberculosis, etc Pain and œdema of legs, jaundice, ascites, not uncommon
5. INGUINAL GLANDS—Œdema of legs

Also lung, spinal cord, and rarely intracranial symptoms

Clinical Types.—

1. CLASSICAL TYPE—Origin, symptoms, and general progress as above. Remissions not uncommon *Death from* (a) Progressive anæmia and exhaustion; (b) Dyspnœa,

from tracheal pressure; (c) Tuberculosis, (d) Sepsis, rarely. *Duration*: usually 2 to 5 years.

2. LOCALIZED TYPE.—One group may enlarge, with no further extension for prolonged period; rapid extension may finally occur. Group may be: (*a*) External, e.g., one side of neck; (*b*) Internal, e.g., mediastinal or retroperitoneal (often with perplexing symptoms). Most chronic form. Rarely, a palpable spleen with anæmia may be sole manifestation (splenomegalic type).

3. ACUTE AND GENERALIZED TYPES.—Rapid course; all glands and lymphoid tissue enlarged.

4. PEL-EBSTEIN SYNDROME. A remarkable relapsing pyrexia. A period of pyrexia of 10 to 14 days occurs, the temperature rising gradually to a maximum of 103° to 105°, and then steadily falling; following this is an apyrexial period of 10 to 14 days. The cycle may recur over many months. There may be malaise and swelling of glands during pyrexia. Retroperitoneal glands practically always enlarged, either alone or with others.

5. LEUKÆMIC TYPHOID TYPE (Ziegler). Onset insidious; malaise, weakness, abdominal discomfort; temperature constantly high or remittent; spleen enlarged; leucopenia. General condition resembles enteric. Retroperitoneal glands enlarged, and perhaps mediastinal, but no external glands.

Diagnosis. From —

1. TUBERCULOUS ADENITIS. Glands tend to be adherent to each other and to skin; ulceration and caseation common; spleen not often palpable. But definite diagnosis only by *removal and examination of a gland*.
2. NEOPLASMS. Sarcoma, lymphosarcoma, lymphoma, etc. Growth rapid, adherent to and infiltrates skin and tissues. Final diagnosis only by histology.
3. LEUKÆMIA.—By blood examination. Distinction difficult (rarely) in early stages of lymphoid leukæmia.
4. SPLENIC ANÆMIA. Spleen very large; severe anæmia; no enlarged glands.
5. SYPHILIS. Glandular enlargement general and slight.

The diagnosis in the rare forms with enlarged internal glands only is very difficult.

Treatment.—

1. MEDICAL. Arsenic has good effect, glands often becoming smaller.
2. X RAYS.—Glands treated become smaller.
3. SURGICAL.—Localized glands should be removed.

Treatment invariably fails finally, and condition is always fatal.

CHAPTER CII.

PURPURA.

The extravasation of blood into the skin. Purpura is a symptom, and not a clinical entity; in some cases a definite disease is present.

Pathogenesis of escape of blood unknown. Change may be in blood or blood vessels; rupture of vessels may be absent, red cells escaping by diapedesis.

Classification.—

1. PRIMARY PURPURA—
   - *a* Purpura simplex
   - *b* Purpura rheumatica — Arthritic purpura
   - *c* Henoch's purpura
   - *d* Purpura hæmorrhagica
2. SECONDARY or SYMPTOMATIC PURPURA
   - *a* SPECIFIC INFECTIOUS FEVERS. Typhus invariable. Frequent in small pox, cerebrospinal meningitis. Severe forms of scarlet fever, measles; rarely enteric; occasionally in other diseases.
   - *b* SEPTIC INFECTIONS. (i) Infective endocarditis, *a frequent and suggestive symptom*. (ii) Septicæmia, pyæmia.
   - *c* BLOOD DISEASES. (i) *Leukæmia, especially acute*. (ii) Aplastic anæmia. (iii) Pernicious anæmia, rarely.
   - *d* TOXIC.—Snake poison, copaiba, quinine, belladonna, iodid.
   - *e* CONSTITUTIONAL AND CACHECTIC CONDITIONS. Chronic nephritis, cancer, tubercle, debility of old age, scurvy.
   - *f* JAUNDICE of any origin if severe.
   - *g* NERVOUS DISEASES.—Rare. (i) Organic: tabes, peripheral neuritis. (ii) Hysteria may be 'stigmata'.
   - *h* MECHANICAL.—Venous stasis, as from paroxysm of whooping cough, epilepsy, or tight bandages.

PRIMARY FORMS OF PURPURA.

In all forms (1) The lower limbs are chiefly affected from mechanical causes. (2) Hæmorrhage into joints never occurs. (3) Symptoms often precede the purpura. (4) The blood shows *a secondary anæmia*. Nephritis is a serious symptom. The spleen may become palpable.

ARTHRITIC PURPURA.

A group of symptoms occurs, one or more of which may be associated with primary purpura, viz.: (1) Pains in joints. (2) Urticaria and erythemata. (3) Gastro-intestinal disturbances. (4) Nephritis and albuminuria. Names have been assigned to various combinations; all intermediate grades occur, the connection being close. There is also a relationship with urticaria and the vasomotor

disturbances or trophoneuroses No hæmorrhage from mucous membranes, or sponginess of gums

Three groups are usually described (1) *Purpura simplex* (2) *Purpura rheumatica* (3) *Henoch's purpura*

Purpura Simplex. Purpura with mild constitutional disturbances Usually in children Legs most frequent site Spots appear in crops Diarrhœa common May be slight joint pains and fever Duration Usually one to two weeks, occasionally six weeks No special tendency to recurrence No sequelæ

Purpura Rheumatica (*Schonlein's Disease**) Association of *purpura with joint pains* Young adult males mostly affected

ONSET Sore throat fever 101° to 103° albuminuria and casts occasionally

CHARACTERISTICS

a RASH *Purpura Urticaria and erythema* often co-exist

b JOINT PAINS *Arthritis* Usually many joints Some swelling, but *never bleeding into joints* May precede purpura

c LIABILITY TO RELAPSES

PROGNOSIS Often protracted for months but rarely fatal

RELATION TO ACUTE RHEUMATISM In purpura rheumatica (*a*) Previous rheumatism rare (*b*) Endocarditis rare (*c*) Salicylates have no effect Also *d* Purpura very rare in typical acute rheumatism

Henoch's Purpura Purpura with colic (Gastro-intestinal disturbances may occur in any purpura)

CHARACTERISTICS

a RASH *Purpura* Urticaria and erythema often co-exist

b ABDOMINAL COLIC Often severe Diarrhœa vomiting, or constipation May be melæna

c LIABILITY TO RELAPSES

Joint pains Nephritis occasionally

Attacks of colic may occur for months before purpura

Appendicitis may be suggested

Intussusception may be simulated very rarely may be present

Cause of colic Serous exudation into intestinal wall, comparable to urticaria, sometimes sero-hæmorrhagic intussusception may commence at site

PURPURA HÆMORRHAGICA.

(*Morbus Maculosus of Werlhof*)

Purpura with severe constitutional disturbance and hæmorrhage from the mucous membranes Usually in girls often with poor health

SYMPTOMS

1 Onset abrupt few days malaise, then —

2 Purpura severe, extensive ecchymose

* Schonlein called his disease 'peliosis rheumatica'. He left no clear account of it.

Purpura Hæmorrhagica—Symptoms, *continued*

3 Hæmorrhage from mucous membranes epistaxis gums less constant hematuria and other sites

Joint pains, vomiting nephritis may occur Fever usual

COURSE —Purpura and hæmorrhages rapid anæmia, progressive weakness Death may occur in few days from weakness Very rarely cerebral hæmorrhage Improvement may commence in one to two weeks

BLOOD —Blood platelets greatly reduced in this, but not other, primary forms Normal expression of serum by contracting blood clot does not occur Coagulation time normal or lengthened to some degree

Purpura Fulminans.—Malignant forms fatal in few hours (Some recorded cases are probably acute leukæmia)

DIAGNOSIS OF PURPURA.

The blood should be examined in every case Diagnosis from Scurvy, no sponginess of gums, no previous abnormal diet Malignant infectious fevers Hæmophilia may be diagnosed in error in purpura hæmorrhagica if purpura absent

TREATMENT OF PURPURA

GENERAL HYGIENE —Rest in bed Light diet *Keep extremities warm.* Correct digestion Stay in bed depends on *sequences* and *albuminuria*

DRUGS Oil of turpentine ♏x to xv, t.d.s Liquor arsenicalis ♏iij, t.d.s, increasing ♏j alternate days Adrenalin (1 1000) locally to mucous membranes Injections of serum (*see* HÆMOPHILIA)

FOR SEVERE LOSS OF BLOOD Transfusion of blood (*see* HÆMOPHILIA p 635)

HÆMORRHAGIC DISEASES OF NEW-BORN CHILDREN.

1. **Traumatic.**—From injury at birth Important after effects Common forms —

a CEPHALHÆMATOMA —Blood between bone and periosteum Absorbed slowly Of little importance unless simultaneous internal hæmorrhage

b MENINGEAL HÆMORRHAGE Usually bilateral May be fatal Probably a cause of porencephaly After effects Spastic paraplegia (Little's disease) idiocy, etc

c STERNOMASTOID After effects Congenital torticollis

Also occurs into abdominal and thoracic organs usually fatal

2 **Constitutional** (*Hæmorrhagic Disease of New born Children*) —Infant usually healthy at birth Hæmorrhage commences in first week. (Blood sucked from cracked nipple may simulate hæmatemesis, etc.)

SITE OF HÆMORRHAGE.—Commonly: (*a*) Mucous membranes; (*b*) Navel; (*c*) Skin; (*d*) Melæna neonatorum.

ICTERUS.—Occurs in most cases: often intense.

In *epidemic hæmoglobinuria* (Winckel's disease), urine contains methæmoglobin; spleen usually palpable.

MORBID ANATOMY.—(1) Ulcers in duodenum or stomach: frequent cause. (2) Syphilitic lesions of liver, etc. (3) May be no changes, even in melæna neonatorum.

PROGNOSIS.—High mortality within few days. Duration rarely exceeds seven to ten days.

TREATMENT. General hygiene. Warmth. Saline infusions. Transfusion of blood has saved cases *in extremis*.

CHAPTER CIII.

HÆMOPHILIA.*

An hereditary abnormality, limited to males but transmitted by females, characterized by a tendency to excessive hæmorrhage and by prolonged coagulation time of blood.

Etiology.—The prototype of hereditary diseases. 'Law of Nasse' holds good: transmitted only by females and exhibited only by males. No full, authentic case in females. cases apparently transmitted by males are explained by wife being of bleeder stock (Bulloch and Fildes). Females tend to great fecundity. In successive generations, percentage of bleeders usually diminishes. Severity of condition varies in different families.

Pathology.—*Coagulation of blood* is delayed, often many times the normal (length depends on method in use). Cause is an abnormality of fibrin formation.

NORMAL CLOTTING is commonly explained as follows:—

MORAWITZ'S THEORY.—Clotting is formation of insoluble fibrin from circulating fibrinogen.

Factors concerned are: (1) Fibrinogen: contained in plasma. (2) Prothrombin: contained in plasma; possibly formed from blood-platelets. (3) Thrombokinase: contained in all cells, e.g., walls of blood-vessels. (4) Calcium.

First Stage.—Prothrombin is converted by thrombokinase, in presence of calcium, into thrombin (or fibrin ferment), which is thus always formed when blood meets other cells.

Second Stage.—Fibrinogen is converted by thrombin into fibrin—i.e., clot forms.

* *See* Bulloch and Fildes, "Hæmophilia", *Treasury of Human Inheritance* (London: Dulau & Co., 1911.). An exhaustive monograph.

Hæmophilia—**Pathology,** *continued.*

CAUSE OF DELAYED CLOTTING IN HÆMOPHILIA. Evidence incomplete, but :-

1. Fibrinogen not at fault, as clot when formed is normal.
2. Calcium is not deficient in amount.
3. Thrombokinase and prothrombin from hæmophilic blood have shown no abnormality when examined by action in clotting other blood (experiments not very conclusive)

Error is probably in formation of thrombin, possibly deficiency of thrombokinase i.e., error fundamentally in tissue-cells and not in blood. Clots may be present in wound, yet bleeding continue.

ESTIMATION OF COAGULATION TIME OF BLOOD

Withdraw blood from vein, to prevent mixture of tissue juices, which will often cause normal clotting. No danger with fine needle. Coagulation time in given individual often varies at different periods.

Symptoms.—Constant liability to excessive hæmorrhage from slight injuries. Usually commences in early infancy, but is *rare at birth* (i.e., hæmorrhage from navel), tendency diminishes with age; also varies greatly at different times.

ONSET AND CHARACTER OF BLEEDING Probably *always trauma*, but often trivial. A slight cut persists in bleeding, dripping like a sponge, not abnormally profuse in rate, but prolonged in time.

SITE OF HÆMORRHAGE may be :-

1. EXTERNAL. Tooth extraction, epistaxis, gums especially. No site, however, is exempt. Cuts, even when trifling, circumcision, etc.
2. INTERNAL. Subcutaneous hæmatomata, often large and spreading, following slight trauma.
3. JOINTS. Few hæmophilics escape. Mainly large joints, especially knee. Bleeding rapid. Blood may be absorbed completely and leave no sequel, or organization and ankylosis may result.
4. SPINAL CORD. - Transverse myelitis may result.

BLOOD.—Subsequently shows *secondary anæmia* if hæmorrhage severe. Blood-platelets are normal.

Diagnosis.—Essential points are : (1) In males only, (2) *Repeated prolonged* hæmorrhages, on *slight* provocation, commencing in *infancy*, (3) Delayed coagulation of blood; also (4) Hereditary, (5) Transmitted by females only. Diagnosis from :

PURPURA HÆMORRHAGICA.—This may be recurrent e.g., after every tooth extraction—and coagulation time may be somewhat prolonged. Arthritis common. Females not exempt. Not hereditary. Blood-platelets very scanty.

Prognosis.—Worst in childhood : improves with age. Severity varies in different families.

Treatment.—

CAREFUL PROPHYLAXIS in susceptible persons.

LOCAL TREATMENT.—Gently wash clot from site. Apply direct cautery, adrenalin (1–1000), or human blood or extract of thymus, to promote clotting.

SERUM INJECTIONS.—Nature of serum: human, rabbit, or horse (commercial sample if unavoidable, but its freshness is important). Method of injection: intravenous safer than subcutaneous (which may cause hæmatoma). Dosage: 10 to 20 c.c., may be repeated. As prophylactic, injection once a month. (Gulland.)

TRANSFUSION OF BLOOD.—For severe loss of blood, direct transfusion of value: 500 to 750 c.c. Donor's blood must be proved to be non-hæmolytic to recipient's.

CHAPTER CII

ERYTHRÆMIA.

(Vaquez's Disease. Osler's Disease. Polycythæmia Rubra.)

A disease characterized by congested appearance, usually cyanosis, by increase in number of red cells, and by enlargement of spleen.

Pathology.—Polycythæmia occurs in various conditions hindering supply of oxygen to tissues: congenital heart disease, emphysema, high altitudes (rarity of atmosphere).

IN POLYCYTHÆMIA VERA.—Bone marrow active, purple colour: hyperplasia of both erythroblastic and leucoblastic tissues: many myeloblasts present. Hence disease considered *a primary hyperplasia of erythroblastic marrow tissue* (corresponding to leukæmia (but more probably secondary).

ENLARGEMENT OF SPLEEN.—May be result of (1) Engorgement by increased volume of blood. (2) Increased hæmostasis necessary. LIVER often somewhat large.

DILATATION OF VESSELS AND ARTERIOSCLEROSIS.—Results from increased volume of blood. Also greater viscosity increases stasis.

Rarely tuberculosis of spleen present.

Etiology.

AGE.—35 to 60 years.

No syphilitic factor. Workers in gas works form high proportion.

Symptoms.

INITIAL COMPLAINTS.—*Headache* and *giddiness*, or *appearance*. Symptoms often slight.

CHARACTERISTICS.—

1. APPEARANCE.—*Plum-coloured*. General congestion all vessels dilated. Lips and ears purple, face brick-red. Or, if cold, general *extreme cyanosis*.

Erythræmia—Symptoms, *continued.*

2. SPLEEN ENLARGED.—Usually to umbilicus; painless, hard; varies in size.
3. BLOOD CHANGES.—
   a. Volume.—Often double normal.
   b. Red Cells.—7 to 12 million per c.mm. Appearance normal, but a few normoblasts always present.
   c. Hæmoglobin.—130 to 160 per cent of normal. Colour index low.

   Viscosity greatly increased. Leucocytes usually increased (20,000); a few myelocytes. Resistance of red cells to hæmolysis normal (tested against salt solutions).

VARIOUS.—Albuminuria common. Blood-pressure high. Hæmorrhages from mucous membranes or under skin, but never severe. Ascites occasionally. Either polycythæmia or cyanosis may be absent.

Diagnosis.—From other polycythæmias, from cyanosis from coal-tar products, from methæmoglobinæmia. Rarely from tuberculosis of spleen.

Prognosis.—No cure, but long duration. Death from: (1) *Cardiac failure;* (2) *Thrombosis,* e.g., cerebral.

Treatment.—For headache and dizziness: Bleeding, 40 to 80 oz.; transient effect, but repeated when necessary. For cyanosis: Oxygen freely. X rays are of doubtful value. Splenectomy inadvisable.

CHAPTER CV.

ENTEROGENOUS CYANOSIS.

(*Methæmoglobinæmia. Sulphæmoglobinæmia*)

A chronic condition of cyanosis due to presence of abnormal hæmoglobin compounds.

Drugs Producing Cyanosis.—Abnormal cyanotic tints can result from: (1) *Potassium chlorate* (methæmoglobin in blood and also in urine from hæmolysis); (2) Certain coal-tar preparations acetanilide, sulphonal, trional (from methæmoglobinæmia), (3) H_2S poisoning experimentally (sulphæmoglobinæmia), and (4) CO poisoning (carboxyhæmoglobin—coal-gas poisoning).

Enterogenous Cyanosis.—Stokvis recognized chronic cyanosis in absence of drugs or disease, and ascribed it to intestinal disturbances. Later, others discovered that hæmoglobin in these cases may exist as (1) methæmoglobin, or (2) sulphæmoglobin: in both conditions the pigment is entirely *intracorpuscular,* no hæmolysis occurring, and the pigment is *absent from the serum and the urine.* The percentage of hæmoglobin affected may be small, and *no dyspnœa* present; but attacks of severe dyspnœa may occur.

Pathogenesis.—*Nitrites* can convert hæmoglobin into methæmoglobin. They are present in excess in the blood, saliva, and urine of these cases, but not in the fæces. Are the undoubted cause of the condition, but origin is not necessarily intestinal Diarrhœa is common. In certain cases bacilli (colon group) have been isolated from blood cultures. (Comparable is Boycott's methæmoglobinæmia in rats produced by Gaertner's bacillus.)

In sulphæmoglobinæmia, similar excess of nitrites present: constipation usual, but no excess of H_2S found.

Note. Sulphæmoglobinæmia is produced by no known chemical compound except H_2S, and this only experimentally: thus workers in sewers exposed to H_2S never show it Sulphonal causes *methæmoglobinæmia* (also hæmatoporphyrinuria).

Tests for Pigments in Blood.—Spectroscopic. Both varieties show a band between C and D, distinguishable (a) By accurate measurements of position of line, (b) By gradual addition of ammonium sulphide methæmoglobin is rapidly changed to hæmoglobin, sulphæmoglobin is unchanged (except by strong solutions)

Methæmoglobinæmia.—

SYMPTOMS *Lead blue colour of lips*, tongue, and skin appearance of being in extremis May be no dyspnœa or symptoms except headache and some weakness but severe dyspnœa may occur Diarrhœa occasionally, stools may be offensive. Clubbing of fingers has occurred

BLOOD Colour, brown Count, normal No pigment in serum or *in urine*.

DURATION Chronic, many years, but depth varies at different periods

Sulphæmoglobinæmia.—As methæmoglobinæmia, but *constipation* usual.

Diagnosis.—*Cyanosis* from other causes, e.g., heart disease. *Methæmoglobinæmia* from effects of potassium chlorate, coal tar products, etc., hæmolysis occurring, the pigment is present in the urine. *Sulphæmoglobinæmia* is invariably idiopathic

Treatment.—*Milk diet* (often reduces tint). Possibly special attention to mouth. *Regulate bowels.*

Section IX.—DISEASES OF THE CIRCULATORY SYSTEM.

CHAPTER CVI.

CARDIAC SOUNDS AND MURMURS.

HEART SOUNDS.

Cause of the Heart Sounds.

THE FIRST SOUND is produced by (1) Closure of mitral and tricuspid valves, (2) Contraction of ventricular muscle in systole (whence prolongation and slight dulling)

THE SECOND SOUND is produced by closure of aortic and pulmonary valves

Normally

At mitral and tricuspid areas first sound louder than second

At aortic and pulmonary areas second sound louder than first

Aortic second sound fainter in youth than pulmonary second, and louder in age

Variations in Heart Sounds.

FIRST SOUND—

FEEBLENESS AND SHORTNESS Suggests muscular exhaustion (when other signs present) Distant heart sounds commonly due to emphysema, thick chest wall, or old age

ACCENTUATION.—In mitral stenosis, also in simple tachycardia of excitement or exertion

(a) In dilatation: accentuated, but clear and short Thin stretched wall vibrating rapidly and also transmitting sound readily

(b) In hypertrophy accentuated, but dull and prolonged Thick muscle contracting powerfully, but vibrating slowly and damping sound.

REDUPLICATION—Mitral and tricuspid valves closing separately—i e, left and right ventricular systole asynchronous

LOUD SHORT SOUND and systolic shock *at apex* in mitral stenosis, may occur with completely calcified mitral valves. Due to impact of blood against firm valves and ring, or, according to Broadbent's theory, to contraction of only half-filled left ventricle.

SECOND SOUND.—

ACCENTUATION.—Increased momentum of reflux blood on valves: either from increased velocity—e.g., high blood-pressure—or from increased mass—e.g., aortic aneurysm.

REDUPLICATION.—Occurs either at base or apex, especially in mitral stenosis. Theories :—

a. At base : Asynchronous closure of aortic and pulmonary valves owing to abnormal relative blood-pressures.

b. Audible at apex and not at base—common in early mitral stenosis : Is of mitral origin ; the mitral valves, opening at onset of ventricular diastole, are checked by adhesion of their edges and vibrate in the blood-stream. The second element of the reduplication is thus due to the *opening* of the mitral valves, and so is not truly a 'reduplicated second sound'.

GALLOP RHYTHM.—Three sounds, with accent on second. Probably reduplicated first sound. Generally sign of serious cardiac failure in a hypertrophied heart.

'MUFFLED' SOUNDS.—In febrile conditions. May or may not develop into murmurs. Also in pericardial effusion.

EFFECT OF INSPIRATION with normal heart.—At beginning the first sound, and at end the second sound, may reduplicate.

INAUDIBILITY OF THE SECOND SOUND AT THE APEX.—Common in later stages of severe mitral stenosis.

CAUSES.—(1) Diminished blood-supply to aorta, and hence feeble aortic recoil (second sound at apex is mainly aortic) ; (2) Enlarged right ventricle and auricle displace the left ventricle, which is chief conductor of second sound.

CARDIAC MURMURS.

Classification of Murmurs.—

(I) ENDOCARDIAL.—
1. PHYSIOLOGICAL.
2. FUNCTIONAL : (*a*) Hæmic ; (*b*) Relative incompetence ; (*c*) Febrile.
3. ORGANIC.
4. CONGENITAL.

(II) EXOCARDIAL.
1. PERICARDIAL.
2. CARDIO-PULMONARY.

Note.—'Endocardial' includes all murmurs produced within visceral pericardium, and does *not* imply origin from endocarditis.

I. ENDOCARDIAL MURMURS.

1. **Physiological Murmurs.***—Certain negligible *soft, systolic* murmurs occurring in apparently healthy hearts. Evidence : (a) No alteration in size of heart ; (b) No impairment of function ; (c) Life unshortened ; (d) No post-mortem changes.

2. **Functional Murmurs.**—Murmurs not due to organic disease of the valves or valve rings. Always systolic.

(a) HÆMIC MURMURS.—

OCCURRENCE.—Frequent in anæmia and allied conditions, e.g., exophthalmic goitre.

* To be diagnosed with caution, only after full physical tests and repeated examinations at long intervals. Formerly included among 'functional' murmurs.

Endocardial Murmurs, *continued.*

SITE.—At base, second left intercostal space, usually about 1 inch from edge of sternum. Less commonly at mitral area.

CHARACTERS.—Always systolic. Localized. Usually soft and blowing. Often variable: disappearing on rest or per contra, on exertion (viz., slowing or accelerating pulse); or on firm pressure by stethoscope.

THEORIES OF ORIGIN (basal murmur).—

(i) Dilatation of pulmonary artery beyond valves, and diminished viscosity of blood (generally accepted).

(ii) Relative incompetence of mitral valve, regurgitation causing vibration in *auricular appendix*, audibility being increased by the retraction of left lung common in anæmia (Balfour). Evidence for: Murmur occurs to left of pulmonary area. Evidence against: Auricular appendix very rarely visible from front of thorax, and when so, at least 1½ inches from sternum (*see below*, NAUNYN'S THEORY).

(b) RELATIVE VALVULAR INCOMPETENCE.—From *muscular dilatation*.

OCCURRENCE.—(1) Severe fevers, severe anæmia. (2) Dilated ventricles; aortic or renal disease, adherent pericardium.

Note.—Even with extreme dilatation there may be no murmurs.

(c) FEBRILE MURMURS.—Frequent in febrile conditions.

CHARACTERS.—*Apical*, systolic murmur, soft, not conducted, pulmonary second sound not accentuated, area of cardiac dullness not increased.

ORIGIN unknown (? contraction of abnormal muscle, or intraventricular currents).

(2) **Organic Murmurs.**—Due to disease of the valves or valve-rings. Systolic, diastolic, mid-diastolic, or presystolic, but position in the cardiac cycle remains constant.

DISTINCTIVE QUALITIES.—(a) Area of maximum intensity, (b) Time in cycle; (c) Direction of conduction.

CHARACTERS.—Rough, or soft and blowing (latter usually regurgitant murmurs). Often '*musical*'. Constant, or change slowly, and little affected by alterations in posture, etc.

MITRAL AREA.—

PRESYSTOLIC MURMUR.—*Mitral stenosis.* Occurs in auricular systole, hypertrophied auricle driving blood through a stenosed orifice. *Site*, at apex beat, or frequently to right of the impulse. *Not conducted*: often localized to small area. *Crescendo*, ending sharply in loud first sound. (Presystolic murmurs also occur in: (a) Aortic incompetence (Austin Flint murmur); (b) Adherent pericardium).

SYSTOLIC MURMUR.—*Mitral incompetence.* Accompanies or replaces first sound. Maximum at beginning, fades off gradually. Due to regurgitation of blood from ventricle to auricle. *Direction of conduction*: into left axilla, often audible also at angle of scapula. *Very rarely* maximum point to right of apex, conducted upwards towards second left space.

Theory of conduction (doubtful).—(1) Into axilla: from disease of posterior valve flap. (2) Upwards to second left space: from disease of anterior valve flap.

Naunyn's theory: Due to vibrations caused in left auricular appendix. Improbable, but may be cause of audibility at scapula.

MID-DIASTOLIC OR DIASTOLIC MURMUR.—Follows the second sound. Maximum at onset, fades off gradually. Occurs in *mitral stenosis*: is sign of advanced narrowing. Presystolic murmur, if present, follows immediately or after short interval.

Cause: Relaxing ventricle in diastole results in flow of blood through stenosed orifice.

AORTIC AREA.—

SYSTOLIC MURMUR.—*Aortic stenosis.* *Loud rough* murmur, maximum at aortic cartilage, and *conducted upwards into carotids.*

Note.—Aortic systolic murmurs are common, and usually due to roughened valves and causes other than stenosis (*see* AORTIC STENOSIS).

DIASTOLIC MURMUR.—*Aortic incompetence.* Soft, blowing murmur, often audible earliest and best to left of sternum; conducted down sternum.

TRICUSPID AREA.—

PRESYSTOLIC MURMUR.—May occur in tricuspid stenosis, but is usually absent.

SYSTOLIC MURMUR.—Soft murmur. Localized, or conducted to right. Occurs in tricuspid incompetence, but diagnosis justified only with concomitant venous pulsation.

PULMONARY AREA.—Murmurs extremely common, but disease of valves rare.

SYSTOLIC MURMURS.—(1) In healthy thin subjects: especially in expiration, in children. (2) Febrile and other rapidly beating hearts. (3) Hæmic murmurs. (4) Cardio-pulmonary murmurs. (5) Pulmonary stenosis and other congenital lesions. Rarely: (6) Mitral incompetence: murmur conducted upwards.

DIASTOLIC MURMURS.—(1) Aortic incompetence. Extremely rare: (2) Pulmonary incompetence. Occasionally: (3) Mitral stenosis, late stages: transient incompetence from high pulmonary pressure (Graham Steell); murmur disappears when tricuspid incompetence occurs.

BRUIT DE DIABLE.—Audible in neck in conditions of low blood-pressure: ascribed to alterations in calibre of the veins in the neck leading to compression on passage over cervical fascia.

4. **Congenital Murmurs.**—Practically always to left of sternum, in neighbourhood of pulmonary area. (See CONGENITAL AFFECTIONS OF THE HEART.)

II. EXOCARDIAL MURMURS.

(1) **Pericardial Murmurs.**—In acute pericarditis.

TIME.—Systolic or 'to-and-fro,' but not corresponding accurately to onset of systole or diastole.

SITE.—To left of sternum, near centre of cardiac dullness; or close to sternum.

CHARACTERS.—Very superficial; grating or creaking character; localized; vary rapidly; often affected by changes of posture and respiration, but *not abolished by holding breath.*

2. **Cardio-pulmonary Murmurs.**—Occur in diseased conditions where heart and lung meet—e.g., adhesions, dilated heart; some due to systole sucking in, and diastole expressing air from, a portion of lung.

SITE.—Usually to left of cardiac dullness; sometimes at base.

CHARACTERS.—Generally late systolic; short; affected by respiration, maximum in unforced inspiration. May be audible in trachea.

DIFFERENTIATION OF FUNCTIONAL AND ORGANIC MURMURS AUDIBLE AT MITRAL AREA.

An *apical systolic murmur* may be: (a) Febrile; (b) From relative incompetence; (c) Organic.

1. **During Acute Fever.**—In absence of other cardiac signs, immediate differentiation of (*a*), (*b*), and (*c*) is impossible: diagnosis depends on subsequent watching.

As temperature falls: (i) murmur subsiding—condition may be (*a*) or (*b*); (ii) murmur more marked—probably (*c*).

Note.—At onset, a murmur may be due to (*a*) or (*b*), and later be due to (*c*), especially in acute rheumatic fever.

2. **Murmur Persists after Apyrexia.**—To decide importance of murmur, consider: (*a*) Size of heart. (*b*) Rate and rhythm: (i) Rapid rate suggests organic murmur; (ii) Slow, suggests functional murmur. (*c*) Response to effort. (*d*) Advance or alteration in murmur over a long period.

CHAPTER CVII.

PERICARDITIS.

Inflammation of the pericardium, simple or suppurative, arising by spread through the blood-stream, by extension from neighbouring organs, or by injury.

Etiology and Classification.—

(1) PRIMARY IDIOPATHIC PERICARDITIS.—Extremely rare. Usually pneumococcal.

(2) SECONDARY PERICARDITIS.– Causes :—

(a) INFECTIVE, TOXIC, PROCESSES CONVEYED THROUGH THE BLOOD-STREAM.—

i. Rheumatic pericarditis.

ii. Pericarditis in pneumonia.

iii. Septic pericarditis: In septicæmia of any origin, especially puerperal fever and acute necrosis of bone. Invariably fatal.

iv. Terminal pericarditis: In chronic nephritis, diabetes, and debilitating conditions.

v. Acute specific fevers: In scarlet fever. In enteric, rarely. Others very rarely.

vi. Tuberculous pericarditis.

(b) DIRECT EXTENSION OF INFLAMMATION.

(c) TRAUMA.—Perforating wounds, fractured ribs, foreign bodies in œsophagus, etc.

Groups i to iv include nearly all the cases

Rheumatic Pericarditis.—Never becomes purulent.

OCCURRENCE.—Most frequent in early life, between ages of 5 and 20. Accounts for nearly all cases of pericarditis occurring at this period. Rare after age of 25. Males and females equally affected.

RELATION TO MANIFESTATIONS OF RHEUMATISM.—In children, arthritis often slight. May occur with acute tonsillitis only; or with chorea, especially when *rheumatic nodules* are present. In adults, generally with severe arthritis. Endocarditis usually present, and in children nearly always.

TIME OF ONSET DURING COURSE OF ACUTE RHEUMATISM.—Variable. In children usually late in attack. May precede arthritis. May occur in first or any subsequent attack; most frequently in first.

Pericarditis in Pneumonia.—Not uncommon complication in pneumonia, bronchopneumonia, and empyema. More common when right lung is affected. Most frequent under age 5; at that period is commonest cause of pericarditis. Dry pericarditis is often not diagnosed (owing to râles obscuring rub). An effusion usually becomes purulent, and is almost always fatal. Organism generally pneumococcus. Condition is usually ascribed to direct extension of inflammation, but may be a common infection through the blood. Thus pneumococcal pericarditis may be primary or may precede lung affection.

Terminal Pericarditis. In nephritis—rare under age of 35. Most frequent in chronic nephritis. Occurs late in disease, shortly before death. Effusion does not become purulent.

Terminal pericarditis may occur in any chronic illness. Is usually not diagnosed, and clinically of little importance.

Tuberculous Pericarditis.—Rare. May be: (1) Primary, either acute or chronic. (2) With pulmonary tuberculosis. (3) General induration of the serous membranes (polyoserohomenitis): probably not always tuberculous.

Pericarditis, *continued.*

Direct Extension of Inflammation.—From disease of neighbouring organs and tissues, e.g., glands, sternum, ribs; rarely neoplasms (sarcoma only). Possibly also in pneumonia.

CLINICAL VARIETIES OF PERICARDITIS.

Three groups may be recognized pathologically and clinically: (1) Acute fibrinous or sero-fibrinous pericarditis; (2) Pericarditis with effusion; (3) Adherent pericardium (chronic adhesive pericarditis).

I. ACUTE FIBRINOUS OR SERO-FIBRINOUS PERICARDITIS.

Morbid Anatomy.—Changes similar to inflammation of other serous membranes. Progressively: (1) Hyperæmia; (2) Loss of lustre of surface; (3) Fibrinous exudation, at first easily removable, increasing in amount. Surface becomes shaggy, 'bread-and-butter' appearance; some exudation of fluid. Amount of fibrin and fluid variable. In 'dry or plastic pericarditis' effusion slight, and adhesions may form rapidly; most common in children. In severe cases myocardium affected.

Symptoms.—Often slight, and condition not diagnosed.

(1) PAIN.—Often absent, especially in young children, rarely severe, not increased by pressure. Thus differs from pain of pleurisy.

(2) FEVER.—Usually present, no special course. In rheumatic form, hyperpyrexia may occur.

In acute rheumatism, pericarditis suggested by dyspnœa, pallor, a pinched expression, and a feeble rapid pulse.

Physical Signs.—

FRICTION SOUND ON AUSCULTATION.—Sole physical sign: pathognomonic when present. Due to rubbing of inflamed surfaces; absent when much fluid present.

SITE.—To left of sternum, near centre of cardiac dullness, or close to sternum.

TIME.—Systolic or 'to-and-fro,' but not corresponding accurately to onset of systole or diastole.

CHARACTERS.—Very superficial; grating or creaking in quality; usually very local. Tends to vary, and often only present intermittently. Often affected by changes of posture and respiration, but not abolished by holding breath. (In pneumonia, may be obscured by râles in lungs.)

ON PALPATION.—Rarely, fremitus.

Diagnosis.—Friction sound pathognomonic: in absence, diagnosis often impossible. Diagnosis from:—

(1) ENDOCARDIAL MURMURS.—Distinguished by characteristics as above.

(2) PLEURO-PERICARDIAL FRICTION.—Common in pneumonia. Murmur greatly affected by respiration. Endopericarditis often simultaneously present.

Also aortic to-and-fro murmur, if rough and heart rapid.

Termination.—(1) Organization of the fibrin, viz., adherent pericardium. (2) Increase of the fluid, viz., pericarditis with effusion. Very rarely, chronic pericarditis, probably tuberculous. Death may occur at any stage from the associated disease.

Treatment.—*See* PERICARDITIS WITH EFFUSION.

2. PERICARDITIS WITH EFFUSION.

Etiology.—Often follows dry pericarditis, forming the second stage, most commonly in rheumatic and septicæmic types.

Morbid Anatomy.—

CHARACTER OF EFFUSION.—May be :—

1. SERO-FIBRINOUS.—Especially in acute rheumatism.
2. PURULENT.—In septic forms, never in acute rheumatism. In pneumococcal infections, large purulent flakes are present.
3. HÆMORRHAGIC.—Rarely in neoplasms, and occasionally tuberculosis.

AMOUNT OF FLUID. Largest in rheumatic type, but is usually small in children; commonly about 300 c.c. (10 oz.).

Symptoms.—*Onset* may be latent, and effusion when moderate remain undiagnosed. The severity and combination of the following symptoms vary greatly :—

1. GENERAL APPEARANCE.—Anxious and pallid, often suggestive.
2. RESTLESSNESS and INSOMNIA.—Common; in severe cases may be extreme. *Delirium* not uncommon; especially occurs with hyperpyrexia.
3. DYSPNŒA.—The most common symptom; increases with amount of effusion, and often becomes extreme. Upper costal breathing marked. Pulse-respiration ratio may reach 2 to 1. Patient most comfortable on left side or semi-recumbent.
4. PAIN.—Commoner in effusion than in dry pericarditis. Varies from sense of tightness to severe pain. Situation precordial, or, in later stages, epigastric. Increased by pressure on sternum. Distribution may be anginal, or in either side from pleurisy. Precordial hyperæsthesia may occur.
5. PULSE.—Rapid, but not distinctive—may be irregular. Pulsus paradoxus is occasionally present (pulse weakens during inspiration).
6. TEMPERATURE.—Not invariably raised. Rarely exceeds 103°. Hyperpyrexia occurs in rheumatic forms.
7. VOMITING.—Occasionally severe.
8. VARIOUS PRESSURE SYMPTOMS.—Irritable cough. More rarely dysphagia, hiccough (affection of phrenic nerve), or aphonia.

Physical Signs (of large effusions).—

INSPECTION.—Marked movements of right side. Cardiac

Pericarditis with Effusion—Physical Signs, *continued.*

impulse wavy or absent. *Rarely*, precordial space bulges—intercostal spaces obliterated and chest wall œdematous. Veins of neck may be distended.

PALPATION.—*Apex beat slight or absent* (disappearance may be watched during accumulation of fluid). Pulsation often in fourth space, produced by wall of right ventricle.

PERCUSSION.—

CARDIAC DULLNESS:—

1. *Note* very dull and sharply defined from lung.
2. *Area increased*, roughly pear-shaped. Marked increase to right in fifth space (Rotch's sign). To left, *extends beyond apex beat.*
3. Increases rapidly with effusion. Shape and extent may vary with posture.

AREA OF DULLNESS AND BRONCHIAL BREATH-SOUNDS at angle of left scapula, and often in axilla, due to compression of lung (Bamberger's sign). Most frequently found in young people.

AUSCULTATION.—

HEART SOUNDS.—*Muffled.* Pulmonary second sound accentuated.

FRICTION SOUND.—Occasionally persists during effusion, especially at base or when patient is erect; often reappears during absorption.

Diagnosis.—Often overlooked owing to the severity of the associated disease. In rheumatic fever is usually easy, in pneumonia very difficult.

FROM CARDIAC DILATATION. — Diagnosis often difficult. Note:—

(1) In effusion: large extent of dullness, with small degree of impulse.

(2) HEART SOUNDS.—(a) In dilatation, sharp and clear; (b) In effusion, muffled.

(3) RIGHT EDGE OF DULLNESS.—(a) In dilatation, roughly parallel to sternum; (b) In effusion, curves concavely to the right (Value doubtful.)

(4) *Compression of lung rare in dilatation*

The nature of the primary disease is the only guide to the character of the fluid.

Course.—Rapidity of accumulation and absorption of fluid varies greatly—a large effusion may form in a few days.

Prognosis.—

1. IMMEDIATE PROGNOSIS.—Depends on primary disease. (a) Sepsis: invariably fatal. (b) Pneumococcal group: prognosis serious. (c) Rheumatic group: fluid never purulent, most cases recover; prognosis varies with degree of dilatation. (d) Tuberculosis: usually chronic, finally fatal.

2. REMOTE PROGNOSIS.—In rheumatic group, in rare cases, there is complete recovery.

Adhesions are constant sequel of pericarditis (*see* Adherent Pericardium).

Treatment of Pericarditis.—

GENERAL INDICATIONS.—(1) Support heart muscle; (2) Allay inflammation. Treatment is directed to: (a) Diminution of heart-beats; (b) Local treatment of the pericardium, (c) Treatment of special symptoms

IN STAGE OF ACUTE PERICARDITIS—

Rest.—Absolute. Posture as patient prefers.

Diet.—Light and nourishing.

Bowels.—Open freely. Salines

Ice-bag to Pericardium, should be applied continuously, weight supported, packed round with absorbent wool to gather condensing moisture. Chest wall must feel cold to be effective. Flying blisters less effectual.

Drugs.—Stimulants are inadvisable.

IN STAGE OF EFFUSION—Continue above treatment, but not too limited diet. Leeches, 3 or 4, should be applied once

SPECIAL SYMPTOMS.—

Restlessness or Insomnia.—Bromide and opium (nepenthe, potassium bromide, or Dover's powder gr x, or injection of morphia).

Vomiting—Alkalis and hydrocyanic acid. Peptonized milk.

Lividity and Respiratory Distress—Oxygen inhalations.

PARACENTESIS, aspiration of the pericardium—

Indications—(1) Effusion large, (2) Dyspnœa and restlessness severe.

Technique.—Insert needle in 4th or 5th left interspace at extreme left limit of cardiac dullness. If fluid is purulent, pericardium must be freely drained

ASSOCIATED DISEASE must be treated—e.g., salicylates in acute rheumatism

CONVALESCENCE—Must be extremely slow. (1) Three weeks' complete rest in bed after pulse and temperature are normal. (2) Further three weeks mainly in bed. (3) Subsequently, four months' careful convalescence before an adult is permitted to return to work.

3. ADHERENT PERICARDIUM.

(*Chronic Adhesive Pericarditis.*)

Pericardial adhesions are a sequel of pericarditis, either acute or with effusion.

There are two types.—

1. Intrapericardial Adhesions.—*Pericardial layers adherent.* Sac obliterated partly or completely. Symptoms often slight or absent even when complete, and physical signs indefinite.

Adherent Pericardium, *continued.*

(3) EXTRAPERICARDIAL ADHESIONS.—Pericardium adherent to surrounding structures, especially sternum; also to lungs, diaphragm, and mediastinal structures. Internal adhesions also present. This is the condition commonly diagnosed as 'adherent pericardium' *Extreme hypertrophy* and dilatation of the heart occurs: weight up to 40 or more ounces.

Causation.—(1) Acute rheumatism: especially in children: predominant cause. (2) Pneumococcal pericarditis. (3) Tuberculosis or generalized serositis rare (*see* CONCATO'S DISEASE, p. 499).

Symptoms.—Latent, as in compensated valvular lesions; or *as in cardiac failure.*

Physical Signs.—

INSPECTION.—The diagnosis depends on inspection, the most characteristic signs being:—

1. ASYMMETRY OF THE CHEST and pericardial bulging due to the extreme enlargement of the heart.
2. CARDIAC PULSATIONS MARKED.—Undulations pass from the third space to the apex.
3. SYSTOLIC RETRACTION in the neighbourhood of the apex beat. Occurs also in cardiac hypertrophy, being caused by systolic contraction of the right ventricle.
4. ADHESIONS TO THE DIAPHRAGM AND OTHER STRUCTURES cause various symptoms: (a) Immobility of the sternum during inspiration (Wenckebach's sign); (b) Systolic retraction in 7th and 8th left interspace in axilla, and 11th interspace posteriorly (Broadbent's sign); (c) Absence of respiratory movements in epigastrium; (d) Diastolic collapse of cervical veins (Friedreich's sign).

PALPATION.—Apex beat is fixed, no change in position on moving patient on side (of little value). Diastolic shock over precordium (very rare).

PERCUSSION.—Area of cardiac dullness greatly increased.

AUSCULTATION.—In rheumatic cases there is usually a systolic murmur of mitral insufficiency, and very frequently a *presystolic murmur.*

PULSE.—The pulsus paradoxus may be present

X RAYS.—May be diagnostic.

Prognosis.—Very bad—young people usually die during strain of puberty. Those survive best in whom physical growth is slight. Prognosis most grave with large hearts and signs of mediastinal adhesions. Death due to cardiac failure.

Treatment.—In general, as for cardiac failure and compensated lesions.

CARDIOLYSIS.—Removal of 4th, 5th, and 6th left ribs. Should be performed in selected cases. Frees the pericardium from the extrapericardial adhesions. The sternum may also be divided above and below area of adhesion.

CHAPTER CVIII.

DISTURBANCES OF THE CARDIAC CONTRACTIONS.*

I. NORMAL AND ABNORMAL CARDIAC CONTRACTIONS.

The Controlling Mechanism of Cardiac Contractions.—

1. INTRACARDIAL.—The contractions are under the control of remnants of the 'primitive cardiac tube'. These form a chain of connections, and are the junctional tissues from the great veins through the auricle to the ventricle. They consist of :—
   - (a) SINO-AURICULAR NODE (Keith and Flack).—Close to orifice of superior vena cava, at termination of sulcus terminalis. Hence connections proceed : –
   - (b) THROUGH THE AURICLE.—On posterior wall and inter-auricular septum. Connecting with :—
   - (c) AURICULO-VENTRICULAR NODE.—On right side of base of inter-auricular septum, internal to orifice of coronary sinus. Whence arises :—
   - (d) AURICULO-VENTRICULAR BUNDLE (Bundle of His).—Fibres have large nuclei, faint striation, and stain pale. The main bundle runs below the interventricular septum, the pars membranacea, and divides into right and left septal divisions. These subdivide, pass to papillary muscles, and form a network communicating with muscle fibres.
2. EXTRACARDIAL.—
   - (a) VAGUS.—Normally inhibits rate of contractions. In complete absence of vagal control, human auricle probably would contract at rate of 150 to 160.
   - (b) SYMPATHETIC SYSTEM.—Supplies accelerator fibres to heart. From first four dorsal segments, through inferior cervical ganglion.

Origin of Normal Contractions.—*Normal stimuli* causing contractions arise in the *sino-auricular node*, hence called the 'pacemaker' (Lewis), and follow the course of the primitive cardiac tissue, stimulating consecutively auricle and ventricle.

Origin of Abnormal Contractions.—These may arise as follows: (1) Stimuli can originate from any portion of the primitive cardiac tissue. (2) Stimuli, either normal or abnormal, may be blocked by disease in the junctional tissues either partially or completely, or by meeting muscle in 'refractory' state. The two types may, and frequently do, co-exist.

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* T. Lewis, *Clinical Disorders of the Heart Beat* (Shaw & Sons). Mackenzie, *Diseases of the Heart* (Oxford Medical Publications).

Cardiac Contractions, *continued.*

Classification of Abnormalities of Contraction.—

(1) VARIATIONS IN VAGUS CONTROL.—
SINUS IRREGULARITIES.

(2) ABNORMAL ORIGIN OF STIMULI.—
EXTRASYSTOLES (premature contractions).
SIMPLE PAROXYSMAL TACHYCARDIA.
AURICULAR FIBRILLATION.
AURICULAR FLUTTER.

(3) INTERFERENCE WITH PASSAGE OF STIMULI.—
HEART-BLOCK.

(4) IMPAIRMENT OF CONTRACTILITY.
PULSUS ALTERNANS.

Group 2 includes abnormalities of very varying importance and symptomatology. (Simple paroxysmal tachycardia is, pathologically, the regular repetition of an extrasystole, but has no clinical relation to it.)

The Functions of Cardiac Muscle (enunciated by Gaskell) -
(1) Stimulus production—rhythmicity; (2) Excitability; (3) Contractility; (4) Conductivity; (5) Tone.

Abnormalities of contractions have been classified also on the basis of these functions (unsatisfactory).

II. PALPITATION.

Consciousness of temporarily abnormal action of the heart.

Note.—'Palpitation' is a symptom present and complained of in many conditions, and not a clinical entity.

Two factors must be present: (1) Consciousness of the heart-beat; (2) Abnormality—in increased force, increased rapidity, or irregularity. Consciousness of *normal* heart-beat in debility is thus excluded.

ABNORMALITY may be:—

REGULAR AND RAPID: e.g., common in emotions, after exertion, etc.; also auricular flutter and paroxysmal tachycardia.

IRREGULAR AND RAPID: e.g., auricular fibrillation.

IRREGULAR, NOT NECESSARILY RAPID: e.g., extrasystoles, common form. Consciousness may refer to (1) the pause, (2) the big beat following the extrasystole.

FORCIBLE, NOT NECESSARILY RAPID: e.g., emotions, hysteria.

Etiology.—

(5) EXCITABILITY OF NERVOUS SYSTEM.—Common type. Emotions; hysteria; neurasthenia; puberty, menopause, menstruation. Da Costa's 'irritable heart' ('disordered action of the heart'). Anæmia and debilitating conditions.

(6) TOXIC.—Acute fevers. Tobacco; tea; alcohol.

(7) GASTRIC REFLEX.—In dyspepsia.

(8) ORGANIC DISEASE OF HEART.—Valvular. Myocardial. Disorders of rhythm. (Palpitations often absent.)

Symptoms.—Complaint varies with the type of abnormality—viz., fluttering, throbbing, or 'heart stands still' (pause in

extrasystoles). Various degrees of lassitude, cardiac distress, mental depression, and fear. When severe: sitting preferred, pressure applied to heart, deep breaths taken.

Duration.—Few minutes to hours. Termination gradual, or, less often (but especially in gastric group), sudden. In neuroses, at cessation, often passage of much pale urine or flatulent eructations.

Physical Signs.—*In neurotic cases* negative: sounds clear and loud: may be hæmic murmurs: vessels often throbbing and dilated.

Prognosis.—Depends on cause. In neurotic group, life not shortened. In toxic group, recovery good with removal of cause.

Treatment.—In youth, sharp walk often terminates attack. Feelings of exhaustion and depression need rest. Treat etiological factor.

III. TACHYCARDIA.

Increased rapidity of the heart occurs in many conditions; it may be continuous or discontinuous; is often associated with irregularities of rhythm.

VARIETIES.—

(1) SIMPLE TACHYCARDIA.

(2) PAROXYSMAL TACHYCARDIA.—(a) Simple paroxysmal tachycardia; (b) Auricular fibrillation (*see* p. 660); (c) Auricular flutter (*see* p. 657).

The essential difference between these groups is that in the first, simple tachycardia, the cardiac impulse starts at the normal site, the 'sino-auricular node'; while in the second, paroxysmal tachycardia, it starts at some other spot. The electrocardiogram alone can prove this: in practice, diagnosis is usually ascertainable by other means.

1. SIMPLE TACHYCARDIA.

VARIETIES.—

1. SIMPLE DISCONTINUOUS TACHYCARDIA.—Rate normal when undisturbed, but response excessive to (a) exertion (due to slight reserve power of heart); (b) emotions; (c) both. Not invariably, but frequently, constitutes 'palpitations'.

2. SIMPLE CONTINUOUS TACHYCARDIA.—Rate continuously increased. Usually, in addition, excessive response to exertion or emotion, as in above, these generally constituting 'palpitations'.

OCCURRENCE.—The most important conditions are:—

(a) ACUTE FEVERS.

(b) DEBILITATING CONDITIONS.—(i) Phthisis (early and important sign). (ii) Result of prolonged pyrexia; especially in convalescence of enteric and influenza. (iii) Anæmia; wasting from any cause; hæmorrhage.

(c) ORGANIC DISEASES OF THE HEART, valvular and myocardial.

(d) EXCITABILITY OF THE NERVOUS SYSTEM.—(i) Emotions;

Simple Tachycardia—Occurrence, *continued.*

hysteria; neurasthenia. (iv) Da Costa's 'irritable heart' ('disordered action of the heart'). (v) Menstruation, menopause, puberty; also in pregnancy. (vi) Reflexes—e.g., gastric, flatulence.

(e) EXOPHTHALMIC GOITRE.

(f) TOXIC.—Alcohol. Tobacco. Thyroid extract. Various drugs.

(g) PHYSIOLOGICAL.—The average adult rate is about 72. In exceptional cases it may be 85 to 90.

CHARACTERISTICS.—

a. Rate rarely exceeds 140.

b. *Rate affected markedly* by exertion or rest, emotion, alterations of posture, by atropine and other drugs. Alteration of posture, standing to lying, may slow heart 20 to 30 beats: normal change not above 10. Slows with rest.

c. *Heart does not dilate.*

d. Attacks of tachycardia begin and cease *gradually*; rate during attack may vary considerably.

e. Entire heart contracts more rapidly, diastole shortened more than systole. Conduction of stimulus from auricle to ventricle is accelerated, viz., *a–c* interval diminished.

f. Peripheral vessels dilated and often throbbing. Pulse tracing shows sharp up-and-down stroke, from relaxation of arterial wall.

g. Electrocardiogram normal. Stimulus *arises at normal 'pacemaker'*, the sino-auricular node.

DA COSTA'S 'IRRITABLE HEART'.—Frequently occurs in young soldiers. Complaints of palpitations, throbbing, etc. Rate *increases abnormally* on exertion; usually above normal during rest. No certain etiological or pathological factor; often previous enteric, dysentery, etc, but also occurs in absence of any illness.

DIAGNOSIS.—Etiological factor usually obvious. For exclusion of paroxysmal tachycardia, *see below.*

2. SIMPLE PAROXYSMAL TACHYCARDIA.

DEFINITION.—"Sudden acceleration of heart-rate in response to new impulses arising from a focus removed from the normal pacemaker, viz., the sino-auricular node" (Lewis). A rare condition. The site of the focus is usually in the auricle or *A-V* node. The auricle and ventricle beat at the same rate.

ETIOLOGY.—Occurs at all ages. Commoner in males. Previous rheumatic fever not infrequent.

Associated diseases.—Mitral stenosis most common. Often none. At autopsy may be myocardial changes: nothing constant.

FACTORS CAUSING ONSET.—(a) Exertion or emotion: usual cause. (b) Gastric disturbance, especially *flatulence.* (c) Rarely, influence of certain postures.

CHARACTERISTICS.—

(a) *Rate* usually 140 to 190. *Rhythm regular.*

(b) *Rate unaffected* by exertion, emotion, alterations of posture, rest, or by atropine. Also *unaffected by digitalis*. Thus the new focus apparently is independent of ordinary nervous controls.

(c) Attack begins and ceases *abruptly*.

d. Character of radial pulse tracing.—(i) Onset abrupt; (ii) Regular during paroxysm; (iii) Terminates suddenly; (iv) Subsequently a few slow beats, then rhythm faster than normal and showing extrasystoles.

e. Electrocardiogram.—Stimulus arises at abnormal focus. Auricles and ventricles beat at same rate.

DURATION.—From a few seconds upwards, but rarely exceeds two weeks.

SYMPTOMS.—In short attacks (seconds or minutes), may be none. In longer attacks, severity of symptoms varies with (*a*) duration of paroxysm, (*b*) rate of beat, and also with (*c*) previous condition of heart, and (*d*) excitability of nervous system. In given individuals, successive paroxysms are in general similar in duration, type, and symptoms.

AT ONSET.—Various cardiac discomforts, from 'fluttering' to palpitations.

AS PAROXYSM PROGRESSES.—(a) 'Anginal symptoms'; all degrees to severest angina pectoris. (b) Gastric symptoms; especially flatulence, nausea, and vomiting. (c) Symptoms of cardiac failure (in prolonged attacks); heart dilates rapidly; veins engorged; pulmonary congestion; liver enlarged and tender; finally œdema.

TERMINATION.—

(a) SUDDEN TERMINATION AND RAPID RELIEF.—The usual result, even with cardiac failure. Rapid recovery. Often much flatus or urine passed.

Rarely fatal from:—

(b) Progressive cardiac failure.

(c) Sudden death. Rare.

DIAGNOSIS.—From:—

a. SIMPLE TACHYCARDIA.—Pulse usually over 160, rate unaffected by exertion, posture, etc.

b. AURICULAR FIBRILLATION.—Rhythm regular.

c. AURICULAR FLUTTER.—By duration, venous pulse tracing, and electrocardiogram.

Rapid pulse, vomiting, and abdominal pain have simulated perforated ulcer.

During quiescent periods, usually, occasional extrasystoles.

PROGNOSIS.—Good, as regards life, if no symptoms or signs of heart disease between attacks.

General prognosis varies with: (a) Frequency, and (b) Duration of attacks; (c) Rate during paroxysm; (d) Response to exertion (reserve power of heart); (e) Age: children and young adults may 'grow out' of attacks.

During a paroxysm, prognosis varies with: (a) Previous history; (b) Duration of attack; (c) Symptoms of cardiac failure—a serious sign, but recovery may occur at any point.

Simple Paroxysmal Tachycardia, *continued.*

TREATMENT.—Symptomatic. For cardiac failure, usual remedies: rest, oxygen, morphia, venesection. Digitalis ineffective.

Attack sometimes terminated by *firm abdominal binder* (which may prevent recurrence); occasionally by vomiting, or by some posture ascertained by patient.

Prophylaxis important: Avoid sudden exertion and exciting causes.

IV. BRADYCARDIA.

When the pulse-rate is diminished in frequency, consider:—

1. Are the rates of the pulse and heart-beat identical?
2. Are the ventricles and auricles contracting at the same rate? (By comparison of apex and jugular pulse tracings.)

VARIETIES OF BRADYCARDIA.—

1. SIMPLE BRADYCARDIA.—All chambers contracting at same rate. Regular. Rate rarely under 40.
2. MISSED BEATS.—Extrasystoles not reaching radial pulse. (*See* EXTRASYSTOLES.)
3. HEART-BLOCK, partial or complete.—Auricle and ventricle beating at different rhythms. Usual cause of *regular* pulse under 35. (*See* HEART-BLOCK.)

Less common or important:—

4. AURICULAR FIBRILLATION OR FLUTTER.—Bradycardia rare. Irregular. Usually tachycardia.
5. SINUS IRREGULARITY.—Bradycardia rare. Irregular.

SIMPLE BRADYCARDIA.

1. PHYSIOLOGICAL.—Especially in tall athletic men. Also pulse slows with age.
2. CONVALESCENCE FROM FEVERS.
3. MYOCARDIAL CHANGES.—Fatty and fibroid hearts.
4. INCREASED INTRACRANIAL PRESSURE. E.g., cerebral tumours, apoplexy.
5. TOXIC.—Digitalis. Lead. Uræmia (possibly increased intracranial pressure). Jaundice.
6. VAGUS CONDITIONS.—Rarely, pressure of tumours, etc., on vagus trunk. Vagus neuritis may be cause of certain forms, e.g., diphtheria, influenza, lead.

Less important or constant.—Pregnancy. Exhaustion. Hysteria and neuroses. Anæmia.

Action of Atropine.—Atropine paralyzes vagus nerve-endings, and will thus differentiate extracardial (e.g., cerebral) and intracardial forms (e.g., heart-block, myocarditis). In former group injection of atropine quickens heart; in latter group it has slight or no effect.

V. SINUS IRREGULARITY.

Periodic irregularity of the entire heart due to irregular rhythm of stimuli from the normal 'pacemaker', the sino-auricular node.

Characteristics.—(1) Rate of contractions alternately increases and diminishes: due to alterations in tone of vagus. (2) Beats are of equal strength. (3) Jugular and radial pulse-tracings show similar changes. Disappears when pulse-rate increases.

Occurrence.—

1. DEPENDENT ON RESPIRATION.—Rate increases with inspiration. Common in children; also in young adults on deep inspiration.
2. INDEPENDENT OF RESPIRATION.—Frequent in treatment with digitalis, with rheumatic heart conditions; also occurs in healthy persons.

Prognosis.—Condition of no importance. Therefore must be distinguished from other types.

VI. EXTRASYSTOLES.

(*Premature Contractions.*)

A response of the heart to a stimulus from some abnormal focus arising before the normal impulse is due.

Etiology.—Especially in elderly men, but occurs at all ages.

ASSOCIATED DISEASES.—Severe cardiac conditions frequent.

(1) RHEUMATIC CONDITIONS.—E.g., mitral stenosis. Previous rheumatic fever in one-third of all cases.

(2) MYOCARDIAL CHANGES.—Hypertrophy or dilatation without valvular lesions.

(3) ARTERIOSCLEROTIC GROUP, CHRONIC NEPHRITIS.—Especially in ventricular extrasystoles.

(4) ACUTE INFECTIONS.—E.g., diphtheria, acute rheumatism.

Occasionally.—Pregnancy. Excessive tobacco. Digitalis.

No recognizable factor in many cases: subjects healthy.

Morbid Anatomy.—No special changes.

Frequency of Extrasystoles in liable subjects:—

DIMINISHED.—By increased rapidity of pulse—i.e., fever, exertion; pressure on abdomen.

INCREASED.—By exhaustion, dyspepsia, tobacco; by standing; by suspending respiration.

Nature of Extrasystoles.—The extrasystole is a premature contraction of the ventricle, and may be also of the auricle, in response to a stimulus from some abnormal focus. Note:—

(1) The stimulus may arise from various portions of the junctional tissues, producing *various types of extrasystoles.*

(2) The stimulus may recur: (*a*) Irregularly ('intermittent'); (*b*) Regularly—e.g., every 4th, 3rd, or 2nd beat (pulsus bigeminus)—producing 'grouped beats', a regular irregularity.

(3) *The radial pulse.*—The extrasystole is usually a small contraction, and frequently does not reach wrist. Hence

Extrasystoles, *continued.*

radial pulse slower than apex beat. May be regular, if extrasystoles regular. Irregularity depends on : (a) Extrasystoles not reaching wrist (some or all). (b) Rhythm of the extrasystoles : (i) Intermittent—pulse irregular ; (ii) 'Grouped beats'.

Types of Extrasystoles.—

(1.) VENTRICULAR EXTRASYSTOLE.—Most frequent. Abnormal stimulus arises in ventricular wall or tissue ; ventricle contracts in response, auricle continuing its normal rhythm.

CYCLE OF VENTRICULAR CONTRACTIONS (APEX TRACING).—(a) Normal beat ; (b) Extrasystole—at interval shorter than normal ; (c) Long 'compensatory pause' ; (d) Normal beat, usually a 'big beat', stronger than normal. Cycle of auricular contractions unchanged.

Explanation of 'compensatory pause' : A stimulus arrives from normal focus (sino-auricular node) while ventricle is in 'refractory period', and it fails to respond ; hence no contraction until succeeding normal stimulus.

VENTRICULAR TRACING.—Interval between the two normal beats is *exactly two normal periods*, with the extrasystole intervening.

VENOUS PULSE TRACING.—(a) Position of '*a*' wave shows normal auricular rhythm ; (b) Abnormal '*c*' wave, corresponding to ventricular extrasystole.

Note.—The ventricular extrasystole often closely coincides with the normal auricular systole, producing a *shock in cervical veins* and large '*a*' wave.

(2.) AURICULAR EXTRASYSTOLE.—Abnormal stimulus arises in auricular tissue ; hence both auricle and ventricle contract.

CYCLE OF AURICULAR CONTRACTIONS (by position of '*a*' wave on venous pulse tracing).—(*a*) Normal beat ; (*b*) Extrasystole—at interval shorter than normal ; (*c*) Interval of normal length *—no 'refractory period' and 'compensatory pause' occurring ; (*d*) Normal beat. Cycle of ventricular contractions similar to auricle.

VENTRICULAR TRACING.—Interval between the two normal beats is *less than two normal periods*, with the extrasystole intervening.

VENOUS PULSE TRACING—(*a*) Abnormal '*a*' wave present—'*a*' waves in similar cycle to ventricular upstrokes ; (*b*) Waves '*c*' and '*v*' follow the extrasystole '*a*' wave in normal sequence. (Rarely, the auricular extrasystole and (previous) normal ventricular systole coincide, producing a large '*v*' wave.)

(3.) NODAL EXTRASYSTOLE.—Very rare. Stimulus arises in a site whence it spreads to both auricle and ventricle which contract simultaneously.

* May be *slightly* prolonged

General Characteristics.—

1. RADIAL PULSE IRREGULAR.—Various types.
2. RATES OF PULSE AND APEX DIFFER.
3. SYMPTOMS.—May be none. Commonly: (a) 'Compensatory pause' felt as a void in chest; (b) 'Big beat' felt as 'palpitation', with its usual phenomena. The extrasystole itself is not recognized.
4. PHYSICAL SIGNS.—*Palpation*: Extrasystole usually palpable at apex. *Auscultation*: Extrasystole accompanied by faint first sound (or murmur if incompetence); occurrence of second sound depends on whether extrasystole lifts the semilunar valves—often absent. Presystolic murmur never occurs.
5. VENOUS AND ARTERIAL TRACINGS.—Differentiate auricular and ventricular types.
6. ELECTROCARDIOGRAM.—Shows stimulus arises in abnormal focus.

Prognosis.—

1. Serious cardiac disease often present; prognosis little influenced by extrasystoles.
2. In absence of obvious cardiac disease, note:—
   (a) Extrasystoles may, not infrequently, continue to healthy old age. In infections and similar groups may disappear.
   (b) Increased work on heart and circulation is negligible.
   (c) Auricular fibrillation and the more serious irregularities are frequently preceded by periods of extrasystoles, but they are sequels only to a *small proportion* of extrasystoles.

CONCLUSIONS: Extrasystoles *per se* are not indications for alteration in the patient's life, but are indications for regular examination for more serious conditions.

Treatment.—Treat any causal factor (e.g., tobacco). Bromides if patient is nervous. Digitalis contra-indicated.

VII. AURICULAR FLUTTER.

A condition in which the auricle contracts regularly at a rate of 200 to 350, and ventricular contractions respond to a certain proportion of the auricular stimuli, often to a half.

Mechanism: The 'Circus Movement' of Auricular Flutter and Fibrillation.*—Normal stimulus causing cardiac contraction commences at sino-auricular node. It may be regarded as dividing into two main waves of excitation which pass round the superior vena cava and meet on the further side of the inferior vena cava; behind each wave is a zone of 'refractory' muscle, when the waves meet, the union of the two refractory zones ends the stimulus 'like the meeting of two prairie fires'. From these main waves, stimuli spread through the auricular musculature like 'the ripple on the surface of a pond into which a pebble is

*See Lewis, *Lancet*, 1921.

Auricular Flutter—Mechanism, *continued.*

thrown'; they regather at the auriculoventricular bundles, and pass to the ventricles.

Phenomena of *auricular flutter* may be summarized thus :—

1. One main wave, possibly in consequence of increased rate or myocardial degeneration, meets a zone 'refractory' from previous stimulus, and can proceed no further.
2. The second main wave is not blocked : on completion of normal course, the muscle ahead is not 'refractory' (as it is normally), and hence the wave can proceed along the course normally followed, but in the reverse direction, by the other main wave. The zone which blocked the first wave has now recovered excitability. Hence this second wave completes the circuit to its starting-point.
3. But further—there is nothing to impede its entry on a second circuit. The 'circus movement' is thus established, and the same stimulus travels endlessly round the course.
4. As this wave travels, it spreads, like the normal wave, through the musculature of the auricle, and provokes contractions—usually 300 per minute.

Phenomena of *auricular fibrillation* are very similar. A similar 'circus movement' is occurring. Differences are :—

1. Rate is much greater—about 450 to 600 per minute.
2. The path is shorter—close to the orifices of the veins.
3. The zone of excitable muscle which the wave enters is in a state of 'partial refractoriness', some fibres being still refractory; and the amount of such 'partial refractoriness' varies in successive circuits.
4. Through this zone the wave chooses the path of least resistance, viz., the most excitable and least 'partially refractory' muscle fibres. Hence its path is zigzag and irregular. It thus differs from 'flutter', in which the zone is completely excitable (or almost so) and the wave proceeds directly and regularly. The secondary ripples similarly have an irregular path.

This striking explanation supersedes the former hypothesis that numerous stimuli were produced at irregular auricular foci.

The zone of excitable muscle or 'gap' which the wave of excitation enters is very narrow, remainder of circle being in refractory state. Circus movement will cease if this gap can be closed, either by (1) lengthening refractory period of muscle, or (2) increasing the conductivity, i.e., rate at which wave advances : In either case wave would thus meet muscle still refractory, and stimulus comes to an end. This is sometimes effected by quinidine (*see* p. 662).

Relation to Certain Other Irregularities.—Closely connected with auricular fibrillation. From simple paroxysmal tachycardia, is separated arbitrarily by auricular rate exceeding 200: distinction justified by different symptomatology and result of treatment.

Etiology.—Mainly in elderly males. Associated with: (1) Arteriosclerosis: most commonly. (2) Rheumatic history: about 25 per cent: may be valvular lesions. (3) Syphilis: occasionally.

Morbid Anatomy.—Doubtful; probably fibrosis of myocardium.

General Description.—

1. AURICLE.—Contracts *regularly*; rate 200 to 350; recognized by venous tracings or electrocardiogram.
2. VENTRICLE.—
   a. Heart-block almost invariable; *usually* 2 : 1 *rhythm.*
   Rate unaffected by exertion or rest: thus differing from healthy rapid ventricular rhythms.
   Less often 4 : 1 and other rhythms, or mixed rhythms producing irregular slow pulses: converted at once to 2 : 1 rhythm by slight exertion. May be complete block.
   Rarely ventricle beats at rate of auricle (ventricular flutter): only transient attacks compatible with life.
   Pressure on vagus or carotid causes transient slowing.
3. GENERAL EFFECT ON HEART.—Condition usually fair. no great dilatation. Ventricular output per beat may be small.
4. DURATION.—May be years.

Physical Signs.—

RADIAL PULSE.—(1) Usually 2 : 1 heart-block; *pulse rapid*, 120 to 160, *regular* in time, also in force, or may be pulsus alternans. Less commonly: (2) 4 : 1 heart-block, etc., or rhythm varying; pulse slower, irregular in time and in force (3) Complete heart-block; pulse very slow and regular.

VENOUS PULSATIONS.—Rapid in all forms.

VENOUS PULSE TRACINGS.—Numerous small '*a*' (auricular) waves; larger '*c*' and '*v*' waves. Recognition often difficult.

ELECTROCARDIOGRAM.—In all forms shows: (1) Auricle is beating regularly; (2) Ventricular contraction is always response to an auricular stimulus—i.e., there are no extrasystoles. This is proof of auricular flutter.

Symptoms.—

1. WITHOUT OTHER SIGNS OF HEART DISEASE.—Symptoms depend on small output of blood during rapid contractions—viz.: (a) Rapid exhaustion and shortness of breath on exertion. (b) Giddiness: fainting common.
2. SUPERVENING IN CHRONIC HEART DISEASE.—Symptoms accentuated severe cardiac failure occurs sooner or later. Effect on heart comparable with auricular fibrillation, but rarely so severe.

Diagnosis and Characteristics.—

1. VENTRICULAR RATE RAPID.—(a) Rapid, regular pulse, 120 to 160, in elderly persons. (b) Unaffected by rest or exertion.

Auricular Flutter—Diagnosis and Characteristics, *continued.*

(c) Under digitalis: slower and irregular. (d) Heart-block almost invariably present—usually 2 : 1.

2. VENTRICULAR RATE SLOW.—Complete or high partial heart-block. Pulse often irregular. Symptoms slight. Diagnosis often missed or impossible without electrocardiogram.

Prognosis.—Depends on associated lesions and reaction to digitalis.

Treatment.—*Digitalis* specifically indicated (for dosage, *see* p. 661).

RESULT OF DIGITALIS TREATMENT.—(1) First, *slows* pulse, auricle, and ventricle. (2) Next, *converts flutter into fibrillation*; *pulse irregular.* (3) Finally, digitalis is stopped: fibrillation ceases and normal rhythm is resumed. Symptoms of failure, if present, improve rapidly. Repeat digitalis if flutter returns. If normal rhythm not restored, keep pulse rate 60–90 by digitalis, and give quinidine.

VIII. AURICULAR FIBRILLATION.

A condition in which the auricular musculature does not contract harmoniously, and consequently fails to expel blood from the auricle.

Mechanism.—*See* AURICULAR FLUTTER.

Etiology.—

AGE.—Two groups: (1) Rheumatic, age 10 to 50; sexes equal. (2) Non-rheumatic, age 40 to 80; commoner in males.

ASSOCIATED DISEASES.—(1) *Predominant is mitral stenosis.* (2) Rheumatic history without mitral stenosis. (3) Elderly group with arteriosclerotic and myocardial changes. (4) In course of exophthalmic goitre. Rarely during acute infections.

Morbid Anatomy.—Usually fibrosis of myocardium, especially in auricles: definite relation yet unproved. Generally valvular lesions, hypertrophy, and dilatation.

General Description and Results.—

1. AURICLE.—When experimentally produced in animals by faradization: auricle permanently in condition of diastole, fine fibrillating movements visible incessantly in walls.
2. EFFECT ON VENTRICLES.—Contractions *totally irregular in time and force*: owing to irregularity of stimuli emerging from auricles, also influenced in rate by impaired conductivity of junctional tissues by co-existing disease. In essential cause of rapid irregular 'mitral' pulse.
3. GENERAL EFFECT ON HEART.—Great additional work leads to dilatation: usually slowly, but in rare paroxysmal fibrillation may occur in few hours.
4. CARDIAC FAILURE.—Follows from above effects.
5. DURATION.—When established, usually permanent until death.

PAROXYSMAL FIBRILLATION.—Rarely attacks are transient. Differs from 'simple paroxysmal tachycardia' only by irregularity of ventricular contractions. Often become permanent.

Physical Signs.—Depend on two factors: (1) Irregularity of ventricular contractions; (2) Absence of auricular contractions.

VENTRICLE.—Complete irregularity in time and force.

AURICLE.—In *mitral stenosis*: (a) *Presystolic murmur disappears* (no auricular systole). (b) Diastolic murmur frequently remains (due to ventricular diastole—*see* CARDIAC SOUNDS AND MURMURS): can be timed in long pauses.

PULSE.—*Rapid*, 100 to 160; complete irregularity; many beats fail to reach wrist. (*Very rarely*: Slow and almost regular, recognition of condition by tracings only.)

VENOUS PULSE TRACING.—Auricular '*a*' wave absent. Ventricular contractions produce waves. Occasionally, fine oscillations due to auricular contractions. Interpretations difficult when great rapidity.

Symptoms.—No symptoms special to auricular fibrillation. May be consciousness of irregularity. Commonly, *usual symptoms of cardiac failure* (angina rare). Occasionally, no symptoms, especially when ventricle beats slowly.

Diagnosis.—Of cases of cardiac failure, 60 to 70 per cent are associated with auricular fibrillation. Is the common cause of *rapid irregularity*, pathognomonic in mitral stenosis with loss of presystolic murmur.

Note. Slow irregular pulse, and diastolic murmur maximal at apex, is auricular fibrillation and not aortic regurgitation.

Prognosis.—Serious. Depends on: (1) Co-existing disease; (2) Severity of associated symptoms, considering the duration of the arrhythmia; (3) Rate of ventricle apart from treatment; (4) Reaction to digitalis, in rate and in symptoms. Great enlargement unfavourable.

DEATH.—Usually as in cardiac failure. Occasionally sudden: probably ventricular fibrillation.

Treatment.—Rest and *digitalis*. Digitalis is specifically indicated. Action: blocks impulses at auriculo-ventricular junction, and thus *slows ventricle*; auricular fibrillation remains. Quinidine sometimes restores normal rhythm.

METHOD OF ADMINISTRATION OF DIGITALIS WITH PATIENT IN BED.—

a. Commence with large doses: tincture ♏ xv to xx three or four times daily.

b. If no reaction in four days, increase until (i) pulse slows, or (ii) nausea, headache, diarrhœa occur (overdose).

c. When pulse slows to 80, omit: recommence smaller doses if rate rises.

d. If 'coupled beats' occur, discard digitalis entirely.

e. In very severe cases: Commence with strophanthin gr. $\frac{1}{100}$ in ʒj normal saline, intravenous injection, three doses at two-hourly intervals. Should slow pulse in 6 to 12 hours.

Auricular Fibrillation—Treatment, *continued.*

Massive Dosage.—Under trial. With cardiac failure, an initial dose of ʒj is good treatment, and may be repeated under careful observation.

2. QUINIDINE: EFFECTS IN AURICULAR FIBRILLATION (Hoffmann, Lewis).—This drug in some cases restores normal rhythm.

MODE OF ACTION (Lewis).—(1) *Lengthens refractory period.* This ends circus movement and restores normal rhythm. But it also: (2) Slows conduction: if this action equals or is greater than last, circus movement continues: accounts for failure in 50 per cent of cases. But it always slows *auricular* rate.

RESULTS OF ADMINISTRATION.—Two groups of cases, about equal number:—

1. *No effect on ventricular rhythm.*—Cases with cardiac failure, or which do not respond to digitalis, are not corrected by quinidine: also many others.
2. *Rhythm becomes normal.* Note:—
   a. Ventricular rate increases as auricular rate slows. Due to (i) lesser grade of heart-block, (ii) quinidine partially paralyzes vagus.
   b. Relapses may occur on ceasing quinidine: controlled on further administration.

DOSAGE.—Gelatin capsules, gr. v t.d.s.: effect in a few days. Smaller dose, gr. iij once daily, for preliminary two days. Or administration on successive days of 5, 10, 15, 20, and 25 grains (daily dosage).

GENERAL CLINICAL EFFECTS.—

1. Ventricular rate is usually increased when normal rhythm is restored.
2. Embolism may occur, from clots driven from auricular appendix on restoration of auricular contraction.
3. Clinical condition and comfort of patient show little improvement on restoration of normal rhythm.

CONCLUSION.—Quinidine should at present be used with caution. A course of digitalis should precede administration.

3. PATIENT CONVALESCENT, OR MILDER FORMS.—Tincture of digitalis, ♏v to x, t.d.s., over prolonged periods: larger doses may be necessary.

4. GENERAL TREATMENT.—Rest. Avoid all exertion. Attend to general health.

General Characteristics.—

1. **Usual cause of rapid irregular pulse.**
2. **Common in severe cardiac failure.**
3. **Mitral stenosis usually present: no presystolic murmur.**
4. **Markedly affected by digitalis, and often by quinidine.**
5. **Prognosis serious.**

IX. HEART-BLOCK.

(Including Stokes-Adams' Disease.)

A condition in which impairment of conduction of stimuli from auricle to ventricle finally results in ventricle contracting less frequently than auricle.

Etiology.—Commoner in males. Occurs at all ages (in young, rheumatic; in old, syphilitic).

CAUSAL CONDITIONS.—

1. RHEUMATIC FEVER.—(a) Acute stages: rare, and transient. (b) Chronic form: *usual cause* (may be associated with mitral stenosis, auricular fibrillation, extrasystoles).
2. SYPHILIS.—Gumma.
3. FIBROID MYOCARDITIS.—Origin may be: (a) Arteriosclerosis; (b) Syphilis; (c) Rheumatism.

Less common:—

4. SEVERE FORMS OF ACUTE INFECTIONS.—Enteric, diphtheria, influenza; others rarely.
5. TUMOURS OF BUNDLE OF HIS.—Rare.

Digitalis in large doses may produce transient heart-block in acute rheumatism and severe infections.

Morbid Anatomy.—*Connecting auriculoventricular tissue* always affected, usually *main bundle of His*.

LESION.—(1) In acute infections, leucocytic infiltration. (2) In chronic conditions, fibrosis or calcification; this often spreads from the 'central fibrous body' below which the bundle passes. General myocarditis usually is present. (3) Gummata, tumours.

Types of Heart-block.—(1) Partial; (2) Complete.

1. PARTIAL HEART-BLOCK.—The contractions of the ventricle result from stimuli arriving from auricle. Varieties and progressive degrees:

(a) PROLONGED *A–V* INTERVAL, but all stimuli pass and ventricle and auricle beat at same rate. Recognized by long *a–c* interval in jugular pulse tracings.

Physical Signs (difficult).—In mitral stenosis, interval between presystolic murmur and first sound. If no murmur, may be reduplication.

(b) OCCASIONAL 'DROPPED BEATS'.—The ventricle fails to respond to a contraction of auricle (no pulse or apex beat). Preceded by progressive lengthening of *a–c* interval. Succeeded by short *a-c* interval and recurrence of cycle.

(c) 2 : 1 RHYTHM.—Ventricle responds to alternate auricular contractions. Common in mitral stenosis.

Physical Signs.—In 2 : 1 rhythm, two thrills and two murmurs to each apex beat.

Rarely, 3 : 1 and 4 : 1 or other rhythms. Physical signs complex.

Types of Heart-block, *continued*

GENERAL CHARACTERISTICS —

(i) Radial pulse and apex beat show similar rate and irregularity

(ii) Rate commonly 40 to 50 irregular or intermittent

(iii) Irregularity stopped by exertion, often by atropine

(iv) Venous tracing shows auricle beating faster than ventricle

(v) Silence on auscultation during radial pauses

(2) COMPLETE HEART-BLOCK —The ventricle and auricle beat with independent rhythms, no effective stimuli reaching ventricle

CHARACTERISTICS —

(i) Radial pulse and apex beat show similar rate

(ii) Ventricular and pulse rate very *slow*, 35 or less *regular*

(iii) Unaffected by exertion or atropine

(iv) Venous tracing shows ventricle beating slower than auricle (about 70) but rhythms independent

(v) Attacks of giddiness, etc

PHYSICAL SIGNS —

Pulsations in cervical veins, obviously more rapid than apex beat, also of varying strength e g, *big wave* when auricle and ventricle contract *simultaneously*

Heart sounds though regular, *vary in loudness* and may be reduplicated—depending on coincidence of contraction of auricle and ventricle

Attacks of Heart-block.—Heart-block thus occurs in all grades from slight partial forms to complete In the usual chronic conditions, some varying degree of partial block (e g dropped beats) is constant or almost constant while *attacks* occur of severer partial or of complete block of variable duration Note that definite cardiac disease productive of symptoms is also practically always present - e g, mitral stenosis, fibroid myocarditis *Danger arises from*

1 Concomitant cardiac disease
2 Period of transition from partial to complete block
3 Periods of excessive slowing of ventricular contractions

Symptoms.—

1. THOSE OF ASSOCIATED DISEASE e g, mitral stenosis
2. THOSE ARISING DIRECTLY FROM HEART-BLOCK

(a) GENERAL CIRCULATORY DISTURBANCES As in other cardiac disease, arising from degree of heart block constantly present Usually slight, being compensated by ventricular hypertrophy, if no associated disease is present

(b) ANÆMIA OF THE BRAIN Produces characteristic results Arises in one of two ways (i) In complete or severe partial blocks, periods occur of further excessive slowing or of ventricular asystole, pulse rate 7 to 30, cause unknown (auricular rate unchanged). (ii) At onset of complete heart-block, a period of ventricular asystole occurs before independent ventricular rhythm commences.

Symptoms :—
Minor degrees : pallor, giddiness.
Unconsciousness usual if : (1) Rate below 20 ; (2) Asystole 3 to 7 seconds.
Convulsions, if asystole 15 seconds. Face, upper limbs ; rarely general. Also cyanosis, congestion, respiration deep and irregular. No micturition or biting tongue. May be repeated attacks, and death in 'status epilepticus'.

Stokes-Adams' Disease.—Applied to syndrome, as above, of (1) *heart-block*, (2) *convulsions* Excludes numerous heart-blocks without convulsions Consists of the severe heart-blocks with temporary excessive slowing or prolonged ventricular asystole.

Prognosis of Heart-block – Heart-block is proof of, and prognosis depends on extent of, myocardial disease. Consider : (1) Condition of heart (bad in mitral disease) ; (2) General cardiac symptoms ; (3) Frequency, duration, and severity of syncopal attacks or convulsions.

In chronic forms, prognosis always serious. *Death* occurs from : (1) General cardiac failure—commonly ; (2) *Suddenly*—from prolonged asystole of 1 to 2 minutes (viz., onset of complete block) ; (3) In status epilepticus

In acute febrile group, prognosis during illness worse, but attacks may subsequently cease permanently.

Treatment.—

IN CHRONIC FORMS. Bed not essential.

MILD DEGREES. No special treatment. Digitalis not contra-indicated, though may increase degree of block.

ATTACKS OF HIGH GRADE. Rest in bed. Treat cause (rheumatism, syphilis).

CONVULSIONS —No treatment effective. Lie down at warning symptoms. Flatulence or gastric disturbances may precede or cause onset.

X. PULSUS ALTERNANS.

A condition in which ventricular beats are alternately strong and weak, although the rhythm is regular. Its importance is the seriousness of the ultimate prognosis.

Auricle and ventricle beat regularly : sequence of contraction normal. Condition is ascribed to impaired function of contractility.

Occurrence.—Two groups :—

(1) SEVERE TACHYCARDIA—e g , auricular flutter. Importance in prognosis slight.

(2) NORMAL HEART-RATE. -

(a) In group of myocardial disease, angina, chronic nephritis. Usually in later life. Most frequently following an extra-systole. *Prognosis* : extremely serious life rarely exceeds two years, even when rhythm occurs for a few beats only.

(b) May occur during digitalis administration. Omit instantly.

Pulsus Alternans, *continued.*

Diagnosis.—Distinguished from regular extrasystoles (pulsus bigeminus) by normal cycle. No special symptoms. Rarely recognizable by finger. In cases of group 2 *a*, should be carefully looked for in pulse tracings, especially following an extrasystole: purposely provoked by exertion, etc., if necessary.

Treatment.—Rest. Avoid all strain.

CHAPTER CIX.

AFFECTIONS OF THE MYOCARDIUM.

I. HYPERTROPHY.

Hypertrophy is the response of the heart to chronic demand for extra work. Results from: (1) Organic cardiac disease, valvular or myocardial; (2) Pulmonary disease; (3) Increased blood-pressure. When hypertrophy reaches its limit and demand continues, dilatation proceeds and cardiac insufficiency occurs.

Morbid Anatomy.—

MACROSCOPIC.—Two types described: (1) Eccentric; cavity normal or enlarged; wall thickened: hence heart enlarged. (2) Concentric; cavity smaller than normal (is a post-mortem effect).

MICROSCOPIC.—Muscle fibres increased in length and breadth. Number probably never increased

Hypertrophy of Left Ventricle.—

CAUSES.—

(1.) LOCAL CONDITIONS OF HEART.—(a) Valvular lesions: aortic lesions, mitral incompetence. (b) Pericardial adhesions—to *extracardial* tissues. (c) Fibroid myocarditis.

(2.) GENERAL CONDITIONS.—(a) Arteriosclerosis. (c) Chronic renal disease. (d) Prolonged muscular exertion.

(3) VARIOUS.—Exophthalmic goitre and chronic nervous palpitations. Various toxins, e.g. tea, tobacco; alcohol doubtful. May occur with pleuritic adhesions. Rare conditions: stenosis or coarctation of trunk of aorta.

Note:—

1. Hypertrophy of other chambers occurs subsequently.
2. Greatest hypertrophy: in pericardial adhesions (20 to 40 oz.), aortic incompetence.
3. Hypertrophy with minimum of dilatation: in aortic stenosis, chronic nephritis.
4. Valvular lesions act mainly by increased intracardial pressure.

PHYSICAL SIGNS.—Definite. (1) Apex beat displaced *downwards* (6th space). (2) Impulse forcible and heaving. (3) At apex, first sound *booming*. (4) At pulmonary area, second sound accentuated. Pulse, full. Blood-pressure, raised.

Hypertrophy of Right Ventricle.—

CAUSES.—

(1) CONDITIONS INCREASING PULMONARY PRESSURE.—(*a*) Mitral valve lesions. (*b*) Chronic lung diseases—e.g., emphysema, fibrosis, bronchitis.

(2) In latter stages of: (*c*) Pericardial adhesions; valvular disease of left side.

(3) Right valvular lesions. Rare

PHYSICAL SIGNS (anterior surface of heart is almost entirely right ventricle).—(*a*) Systolic (positive) pulsation in epigastrium. (*b*) Apex beat diffuse (right ventricle). Usually: (*c*) Venous pulsation in neck marked; tricuspid first sound accentuated.

Hypertrophy of Auricles.—*Dilatation* invariably co-existent.

RIGHT AURICLE.—Increased pulmonary pressure. Usually follows right ventricle.

PHYSICAL SIGNS.—Dullness to right of sternum. Venous pulsation in neck.

LEFT AURICLE.—Mitral lesions. Suggested in mitral stenosis by loud presystolic murmur. Diagnosis by X rays only.

Symptoms of Hypertrophy.—Often none during compensation. Occasionally: giddiness, flashes of light, headache, failing memory, shortness of breath.

II. DILATATION.

Dilatation of the heart applies to enlargement of the chambers. Causes are: (1) Conditions which lead to overfilling of a chamber, (2) Conditions which weaken the walls. In general, causes are those of hypertrophy, dilatation resulting when hypertrophy reaches its limit and demand for further work continues unsatisfied: cardiac insufficiency thus develops.

Normally, a heart when increasing its rate shortens diastole; hence relaxation is less complete, and heart *becomes smaller*.

Etiology.—Two causal factors: (1) *Increased intracardial pressure*; (2) *Impaired resistance of cardiac muscle*. May co-exist.

1. INCREASED INTRACARDIAL PRESSURE.—
   a. All *valve lesions*.
   b. Functional disturbances—e.g. emotion, exophthalmic goitre. An ill-defined group.
   c. Acute dilatation from exertion—e.g., dilatation of right heart after sprinting. Recovery rapid: capacity for recovery increased by judicious 'training'.
2. IMPAIRMENT OF CARDIAC MUSCLE.—
   a. Chronic myocarditis—e.g., fibrosis, fatty heart. Hypertrophy may have reached its limit, then dilatation proceeds uncompensated—e.g., in chronic nephritis.

Dilatation—Etiology, *continued.*

b. Acute pericarditis: often serious.
c. Acute endocarditis.
d. Acute fevers: occasionally rapid death.
} Acute inflammations: interstitial and degenerative myocarditis.
e. Pericardial adhesions—mechanical interference with muscle.
f. Anæmia—impaired nutrition of muscle.
g. Disturbances of rhythm—e.g., sometimes in paroxysmal tachycardia, auricular fibrillation.

Heart-strain'; 'Broken Wind'; 'Overstress of Heart'.—Prolonged exertion in those unfit or untrained may cause dilatation and cardiac distress. Immediate recovery may appear complete, yet subject subsequently be permanently unable to undertake exertion. Pathology unknown.

III. DISEASES OF THE MYOCARDIUM.

Classification.—

1. ACUTE LESIONS.—(1) Acute myocarditis: (*a*) Parenchymatous degeneration; (*b*) Interstitial myocarditis. (2) Anæmic necrosis. (3) Embolus and thrombosis of coronary arteries (4) Septic infarcts.
2. CHRONIC LESIONS.—(1) Chronic interstitial myocarditis (fibroid heart). (2) Fatty heart; (*a*) Fatty degeneration; (*b*) Fatty infiltration.
3. VARIOUS DEGENERATIONS.—(1) Brown atrophy. (2) Fragmentation and segmentation. (3) Amyloid degeneration (4) Zenker's degeneration. (5) Calcareous degeneration.

1. ACUTE LESIONS.

1. **Acute Myocarditis.**—

ETIOLOGY.—(*a*) Acute fevers, especially *diphtheria*, typhoid, and sepsis. (*b*) With acute endocarditis, e.g., *in acute rheumatism*. (*c*) With acute pericarditis. (*d*) Intoxications: e.g., in acidosis (as in eclampsia) parenchymatous degeneration often advanced.

VARIETIES AND MORBID ANATOMY.—

(*a*) PARENCHYMATOUS DEGENERATION.—
Macroscopic: Pallor and softness of muscle.
Microscopic: Granular degeneration of muscle fibres.

(*b*) INTERSTITIAL MYOCARDITIS.—
Macroscopic: Nil.
Microscopic: Interstitial tissue infiltrated with small round cells and leucocytes; muscle fibres degenerated.
Probably *chronic* interstitial myocarditis often follows.
Of special importance in acute peri- and endocarditis.
In rheumatic forms, bundle of His possibly affected.

SYMPTOMS.—Indefinite. Affection of muscle suggested (e.g., in diphtheria) by: (*a*) Pulse feeble: easily accelerates. (*b*) Apex beat and heart-sounds feeble; may be soft apical systolic murmur. (*c*) Cardiac dullness slightly increased.

2. **Anæmic Necrosis** (*Acute Necrosis or Softening, White Infarct*).
ETIOLOGY.—Embolus or thrombus of terminal branch of coronary artery.
MORBID ANATOMY.—
Macroscopic.—Irregular wedge-shaped yellowish area; projects above surface. Common site: left ventricle (anterior coronary artery).
Microscopic.—Necrosis of fibres, leucocytic infiltration, gradual fibrosis.
Aneurysm or rupture of heart may result: rare.

3. **Embolus or Thrombosis of Coronary Artery.**—*Sudden death* usually follows sudden blockage of one artery: commonly previous disease present from arteriosclerosis or obliterative endarteritis. If blockage gradual, other artery may supply circulation.

4. **Septic Infarcts.**—In pyæmia, septic emboli may result in multiple abscesses: may rupture into cavity or pericardium.

2. CHRONIC LESIONS.

1. **Chronic Interstitial Myocarditis** (*Fibroid Myocarditis, Fibroid Heart*).
ETIOLOGY.—*Disease of the coronary arteries is the predominant cause* of generalized form (*see* ARTERIOSCLEROSIS) Summary of factors: (*a*) Arteriosclerosis; (*b*) Renal disease; (*c*) Syphilis; (*d*) Old age.
(*Localized* chronic changes may follow acute myocarditis - anæmic necrosis.)
MORBID ANATOMY.—
Macroscopic.—Muscle tough: hypertrophy usual. Frequently, patches of fatty degeneration under endocardium.
Microscopic.—Muscle fibres necrosed and degenerated: excess of fibrous tissue.
Condition most advanced at apex of left ventricle.
SYMPTOMATOLOGY.—Complex. Symptoms are due to cardiac insufficiency, or to abnormal rhythms resulting from fibrosis of stimulus-producing or junctional tissues (*see* DISTURBANCES OF THE CARDIAC CONTRACTIONS).
a. LATENT.—May be no complaints, sudden death occurring.
b. INITIAL SYMPTOMS attracting attention:—
Shortness of Breath.—Most frequent. Slight, or all grades to 'cardiac asthma'.
Dizziness.—Attacks of syncope; *fainting and cold sweats.* Headache. Flashes of light. Memory failing; grades of mental disorders up to (rarely) mania. Epigastric fullness.
Cardiac Pain.—From precordial pain to severest angina.
Extrasystoles.
Heart-block.—All grades to typical Stokes-Adams' disease.

Chronic Lesions—Interstitial Myocarditis, ***continued.***

c. SYMPTOMS OF GENERAL CARDIAC FAILURE.—This results from dilatation or abnormal cardiac rhythms, e.g., auricular flutter.

PHYSICAL SIGNS.—Often indefinite.

PULSE.—Frequently slow, often irregular rhythm. Very variable, depending on: (i) Abnormal rhythms—extrasystoles, heart-block; (ii) Dilatation—pulse rapid.

AUSCULTATION.—Mitral first sound roughened; aortic second sound accentuated.

ARTERIES.—Usually thickened, blood-pressure high, and heart hypertrophied.

2. **Fatty Heart.**—

a. FATTY DEGENERATION.—Common.

ETIOLOGY.—Occurs in: (i) Impaired nutrition: wasting; cachexia from any cause; old age. (ii) Pernicious anæmia: rapid and advanced. (iii) Phosphorus poisoning: rapid and advanced. (iv) With most myocardial changes in varying degree (acute infectious fevers, hypertrophy, myocarditis).

MORBID ANATOMY.—

Macroscopic.—Heart large, flabby, friable.

Microscopic.—Rows of fat globules within muscle fibres May be general or local, latter especially below endocardium. Left ventricle most affected.

SYMPTOMS.—As in chronic interstitial myocarditis. *Latency* common even with advanced changes: death from causal factor.

b. FATTY INFILTRATION.—Invariable in obesity.

MORBID ANATOMY.—

Macroscopic.—Fat accumulates first below pericardium, spreads through wall.

Microscopic.—Great masses of fat cells. Muscle fibres atrophied. Fatty degeneration frequently co-exists.

SYMPTOMS.—Response to exertion limited; pulse soft, regular; heart-sounds faint. Usually no symptoms until dilatation and failure.

3. VARIOUS DEGENERATIONS.

These are of little clinical importance.

1. **Brown Atrophy.**—Common in old age, chronic valvular disease. Heart tough, dark brown. *Microscopic*: Granules of brown pigment near nuclei of muscle fibres.

2. **Fragmentation and Segmentation of Muscle Fibres.**—Occasionally occurs with other myocardial changes.

3. **Amyloid Degeneration.**—Rare. Affects connective tissue.

4. **Zenker's Hyaline Degeneration.**—Mainly occurs in enteric fever.

CHAPTER CX.

ENDOCARDITIS.

Inflammation of the lining membrane of the heart. Usually confined to the valves.

Classification.—Endocarditis may be: (1) Acute: (a) Simple; (b) Infective. (2) Chronic. All variations occur between (a) simple and (b) infective, but in general the distinction is marked, and the progress and prognosis differ greatly. Thus: *Simple*: symptoms cardiac or result of cardiac lesions. *Infective*: symptoms generalized, septicæmic and pyæmic. Hence endocarditis is considered as follows: —

1. SIMPLE. —
   a. ACUTE.—Characterized by vegetations on valves.
   b. CHRONIC. Fibrosis causing abnormalities of the valves: (i) Secondary to simple acute endocarditis; (ii) Secondary to high blood-pressure—no acute stage.
2. INFECTIVE.

Synonyms.—

SIMPLE. —Benign. Verrucose. Warty. Rheumatic.

INFECTIVE.—Malignant. Ulcerative. Septic. Subacute bacterial.

I. ACUTE SIMPLE ENDOCARDITIS.

Characterized by vegetations on the valves.

Etiology.—Probably invariably secondary to some infection: —

1. Acute rheumatic fever
2. Chorea
3. Tonsillitis

} Group of rheumatic affections forming predominating cause of simple endocarditis.

Less commonly:—

4. Specific fevers: especially scarlet fever; occasionally enteric; rare in others.
5. Pneumonia.
6. Phthisis.
7. Terminal in debilitating conditions: cancer, gout, diabetes, nephritis.
8. Fœtal.

NOTES ON ABOVE CAUSES.—

1. *Acute Rheumatic Fever.*—Endocarditis occurs:—
   In one-third to one-half of cases, at least.
   More commonly in children, viz., under 20.
   More commonly in first attack.
   Without definite relation to severity of arthritis.
   Early in attack: usually physical signs by second week.
   Commonly affects mitral valve.

Acute Simple Endocarditis—Etiology, *continued.*

2. *Chorea.*—Usual cause of fatal acute endocarditis. In fatal chorea, almost invariable. Of non-fatal cases of chorea, half develop cardiac lesions.
5. *Pneumonia.*—Infective form commoner than simple.
6. *Phthisis.*—In 5 per cent of autopsies. Probably not due directly to *B. tuberculosis.* No clinical importance.

TRAUMA.—Signs of endocarditis have followed trauma of chest: probably endocarditis was present previously.

No evidence that syphilis causes acute endocarditis.

Recurrent endocarditis on old affected valves common.

AGE.—Onset common in childhood, and especially young adults; rare after 40 years; rare in infants (pericarditis more frequent)

FŒTAL ENDOCARDITIS.—Occurs with or without congenital abnormalities, in the former case being either cause or sequel Nearly always right side, especially pulmonary valves Incidence here ascribed to: (1) Higher pressure in right ventricle; (2) Frequency of abnormalities; (3) Infected blood from placenta.

Morbid Anatomy.—

SITES AFFECTED.—

1. Left heart in great majority. Ascribed to high blood-pressure. Possibly also greater oxygenation of blood.
2. Mitral valve commonest. From greater tension in closure of valve.

MACROSCOPIC.—*Minute vegetations on valves*: irregular warty appearance; may have narrow pedicles Occasionally larger cauliflower growths.

SITUATION.—At site of maximum closure, viz: *Mitral valves*. on auricular surface, short distance from margin. *Aortic valves*: on ventricular surface, from corpora Arantii, following the lunules.

Hyperæmia of neighbouring endocardium rare

HISTOLOGY.—(1) Earliest—degeneration of endothelium (nuclei fail to stain); (2) Then fibrin and leucocytes deposited from blood, forming vegetation; (3) Organization into fibrous tissue follows, by proliferation of endothelial and subendothelial cells; (4) Subsequent cap of fibrin and leucocytes. Micrococci common in fibrin or on surface.

MYOCARDIAL INFLAMMATION in severe cases

SUBSEQUENT CHANGES.—May be either:—

(1) VEGETATIONS ABSORBED and valve becomes normal (not proved).

(2) PROGRESSIVE FIBROSIS OF THE VALVE, causing thickening, irregular contraction, and deformity: valves shorten, edges often adherent to each other. Chordæ tendineæ and papillary muscles affected and shortened. Deposition of lime salts. Such changes (chronic endocarditis) prevent function of valves, and constitute the danger of endocarditis.

(3) INFECTIVE ENDOCARDITIS occasionally follows.

(4) RECURRENT ENDOCARDITIS.—Small recent vegetations on fibrosed valves.

Symptoms.—*None characteristic*: attack often latent and unrecognizable. Most constant is fever. Palpitation sometimes marked.

IN RHEUMATIC FEVER. Suggested by: (1) Increased fever without increased arthritis; or (2) Persistence of fever after subsidence of arthritis; may be (3) Rapid pulse or irregularity; less often palpitation, cardiac pain (probably of myocardial origin).

IN RECURRENT ENDOCARDITIS.—Often prolonged pyrexia, 100° to 102° F.

Physical Signs.—

AUSCULTATION.—Earliest sign: slight roughening or prolongation of first sound: may increase (after several days) to faint murmur. Often reduplication and accentuation of pulmonary second sound.

In acute rheumatic fever, such murmur at apex (or sometimes maximum nearer sternum) is frequently mitral endocarditis; if basal, less definite evidence; if aortic diastolic, undoubted endocarditis.

INSPECTION. Precordial impulse often increased and wavy.

Diagnosis.—Difficulty due to: (1) Absence of symptoms; (2) The fact that a murmur in fever is not proof of endocarditis. Note: (a) Pyrexia, in relation to arthritis or other cause (*see above*); (b) Auscultation; (c) Cardiac symptoms.

IF MURMUR PRESENT:—

1. Is it exocardial or intracardial? (*See* p. 6[illegible].)
2. If intracardial, is endocarditis present? Note:
   a. Myocardial murmurs: Usually some *dilatation*, pulse rapid, and some irregularity, and often dyspnœa. Immediate diagnosis often impossible.
   b. Uræmic murmurs: Soft and blowing, usually at base.
3. If endocarditis is present, is it recent, old, or recurrent? If old: often previous chorea or rheumatism, history of dyspnœa, etc., signs of hypertrophy or dilatation.

Complications.—Rarely cause symptoms.

MYOCARDITIS.—Some degree rarely absent; in rare severe grades, cardiac symptoms present with irregular pulse.

PERICARDITIS.—Not uncommon in children.

EMBOLI.—Uncommon in acute simple endocarditis.

Course and Prognosis.—

1. IN ACUTE STAGE.—Death may occur: (*a*) In chorea: (*b*) Occasionally from pericarditis and myocarditis; (*c*) From rheumatic fever without other obvious cause (rare).

   COURSE SUBSEQUENT TO ACUTE STAGE:—

   a. Symptoms and signs disappear; no recurrence.
   b. Symptoms disappear, physical signs remain. Often good health for years. Usually, after variable interval, recurrent attacks or cardiac failure.

Acute Simple Endocarditis, *continued.*

2. REMOTE PROGNOSIS (impossible in acute stage). —Depends on: (a) Age: worse in children. (b) Degree of valvular affection and myocarditis. (c) Valves involved: aortic worse than mitral; serious if both valves. (d) Opportunities for rest; general health.

Treatment.—*Prolonged rest* is first essential. No treatment in rheumatic group prevents onset. Salicylates of no effect.

If present or suspected, e.g., in rheumatic fever: (a) Rest in bed, complete: two to three months. (b) Blisters, 1 in. diameter, over heart (liquor epispasticus). Ice-bag to precordium, if pulse rapid or cardiac disturbance.

Diet: Avoid meat and meat extracts.

Treat concomitant rheumatic fever.

Digitalis inadvisable except with certain irregularities of pulse.

If restlessness, give morphia.

Careful convalescence. —Iron and arsenic tonic.

II. CHRONIC SIMPLE ENDOCARDITIS.

Sclerosis of the valves leading to various deformities, causing interference with function.

Etiology.—

1. Sequel to acute endocarditis, especially rheumatic.

Result of high blood-pressure, without previous acute attack. Factors (a) Prolonged muscular strain, (b) Advancing age, (c) Alcohol, syphilis, gout, and lead arteriosclerotic influences.

Mitral valve commonest, then aortic. Right side very rare.

Many cases probably rheumatic, even when no history obtainable.

Morbid Anatomy.—

EARLY STAGE.—Valve slightly opaque and thickened, especially near edge at line of maximum closure. May be nodules, but no definite vegetations.

PROGRESS.—Fibrosis advances, tissue contracts, whence valves thickened and deformed, adherent to each other, edges curled.

RESULT IN AORTIC VALVES.—Incompetence, often without stenosis.

RESULT IN MITRAL VALVES.—(1) Valve ring affected and narrowed. (2) Lime salts deposit and form hard ring. (3) Chordæ tendineæ affected—thicken, contract, and shorten: may be extreme. (4) Papillary muscles may fibrose. Hence, with incompetence, usually some grade of stenosis.

Acute endocarditis, with vegetations, often superimposed. Infective endocarditis not uncommon.

Symptoms, Physical Signs, etc.—*See* CHRONIC VALVULAR DISEASE, p. 679.

III. INFECTIVE ENDOCARDITIS.

(*Malignant or Ulcerative Endocarditis*)

The condition is practically a septicæmia, with the focus in the heart. Manifestations vary widely. Characterized by: (1) General septicæmic and pyæmic symptoms; (2) Emboli, with mechanical and suppurative effects; (3) Locally in the heart, destruction of tissue, and various signs and symptoms. Micro-organisms may be present in the blood.

Etiology.—

PRIMARY.—No previous known injury of valves, and no obvious septic focus. Rare.

SECONDARY. Almost always so: secondary either to:

(1) OLD SCLEROTIC VALVES i.e., previous endocarditis common form; or to.—

(2) SEPTIC FOCUS. Common examples are. (*a*) Pneumonia; (*b*) Osteomyelitis, otitis media, puerperal septicæmia; (*c*) Specific fevers.

Rare in acute simple endocarditis of chorea and rheumatism. Not uncommon in congenital lesions.

Morbid Anatomy.—

IN THE HEART.—All gradations from simple endocarditis occur, but in definite cases:

(1) LOSS OF TISSUE IN VALVES. Greater and of wider area than in simple form. *Results*: Aneurysm or perforation of valve; rupture of chordæ tendineæ; perforation of septum or, rarely, of heart.

(2) THROMBUS FORMATION from blood on affected areas often marked.

(3) MURAL ENDOCARDITIS more frequent than in simple form (from infection by contact with valves).

Usual Sites: Left interventricular septum; posterior wall of left auricle.

Changes result in fungating area with large vegetations readily detachable to form emboli.

ASSOCIATED LESIONS.—

1. FROM PRIMARY DISEASE—i.e., septicæmia, pneumonia, etc.
2. EMBOLI—*See* SYMPTOMS. Frequent.
3. MYOCARDITIS.

Complications.—Pneumonia, pleurisy, pericarditis, meningitis: origin either embolic or septicæmic. Enlargement of spleen or liver, and nephritis, may occur from sepsis.

Bacteriology.—Blood cultures to be taken in all cases, presence of organisms constituting the only absolute proof of the disease. Commonest organism is a streptococcus growing in short chains. Others occurring are staphylococcus, pneumococcus, rarely *B. influenzæ*, Friedländer's bacillus, and various other bacteria.

Infective Endocarditis, *continued.*

Symptoms and Physical Signs.—Very variable. Also in given case both symptoms and physical signs vary rapidly. Referable to three classes: (1) *Septicæmia*; (2) *Cardiac*; (3) *Emboli*.

1. SEPTICÆMIA.—
   a. PYREXIA.—High, 103° to 105°. Types: (i) Marked irregularity; (ii) Remittent or intermittent e.g., daily 98° to 104°. (iii) Continuously high (rapid course).
   b. PROFUSE SWEATS.
   c. PROGRESSIVE WEAKNESS: finally delirium.
   d. ENLARGED SPLEEN and, less commonly, liver.
   e. RASHES.—Common. *Purpura*, petechiæ or large hæmorrhages.

   VARIOUS. Albuminuria frequent. Occasional slight jaundice. Diarrhœa, nephritis (occur apart from emboli).
2. CARDIAC.—*Murmurs* and physical signs very variable, and often alter *from day to day* with progress of ulceration and with growth and detachment of vegetations. May be: (a) *Rapidity* or irregularity of pulse; cardiac dilatation, usually but not always with murmurs (especially mitral); signs of cardiac failure. (b) No symptoms or signs
3. EMBOLI.—May be multiple. Often diagnostic. Results are (a) mechanical, (b) suppurative.

   Common Sites.—
   Spleen: Pain in side.
   Kidneys: Pain and hæmaturia; may be palpable
   Brain: Paralyses, aphasia, etc. (*See* CEREBRAL EMBOLISM AND ABSCESS.)
   Retina: Retinal hæmorrhages, sight impaired; optic neuritis.
   Intestines: Diarrhœa; intestinal obstruction
   Lungs, rarely (in right heart affections).

   Note.—It is frequently difficult to be certain whether symptoms are septicæmic or embolic in origin—e.g., enlarged spleen, hæmaturia, purpura.

Clinical Types.—Four types are recognized: (1) *Septic*; (2) *Typhoidal*; (3) *Cardiac*; (4) *Cerebral*. Also *Subacute bacterial*.

1. SEPTIC.—Characteristics: (a) Septic focus present. (b) Symptoms of septicæmia prominent: rigors, sweats, irregular pyrexia. (c) Cardiac symptoms usually absent: may be emboli. (d) *Duration*: rapid, few days.
2. TYPHOIDAL.—Characteristics: (a) Persistent high temperature; marked prostration; diarrhœa; sweats; hæmorrhagic rashes common. (b) Cardiac symptoms absent or various. (c) Typhoidal state; delirium and coma develop. (d) *Duration*: two to four weeks.
3. CARDIAC.—Characteristics: (a) Severe pyrexia and endocarditis, usually with *previous chronic valvular disease*, commonly aortic. (b) Cardiac phenomena marked. (c) *Emboli* very

common. Onset suggested by high fever, weakness, and anæmia, with cardiac physical signs varying frequently, proved by emboli and petechiæ (*d*) *Duration* may be prolonged, many months.

4 CEREBRAL —Resembles meningitis; early delirium or coma. Not common.

SUBACUTE BACTERIAL ENDOCARDITIS.—Is a chronic form of the cardiac type Also known as chronic ulcerative endocarditis *Duration* up to two years. Usually in young adults Previous rheumatic history or evidences of endocarditis in a considerable proportion, but may be none

BLOOD CULTURES -Short chain streptococcus common, low pathogenicity Often sterile

MORBID ANATOMY --Vegetative endocarditis, often extensive and mural Little ulceration

ONSET —Insidious. Anæmia, slowly progressive, producing pallor with some pigmentation of face Weakness and loss of weight and slowly progressive Leucocytosis unusual

FEVER No characteristic type Often daily rise (98°–104°) over many months, with sweats or early rigors Range may be lower Apyrexial periods of weeks may occur

CARDIAC MANIFESTATIONS —May be none, or evidence of old endocarditis No rapid changes

CLUBBING OF FINGERS common

The above symptoms often persist many months and diagnosis very difficult The following tend to develop sooner or later and must be examined for repeatedly: (1) *Osler's nodes Transient, red, tender erythematous spots, especially near finger tips* (2) Purpuric spots and petechiæ of skin (3) Splenic enlargement (moderate in degree) (4) Emboli (5) Nephritis i e, blood and casts found microscopically, without symptoms

TERMINATION -Fatal

Diagnosis --Often difficult Presence of organisms in the blood is the only absolute proof. Most important signs are (1) Emboli; (2) Petechiæ (often earliest diagnostic sign), (3) Changing cardiac signs, (4) Blood culture, (5) Leucocytosis almost invariably marked, (6) Septic focus or chronic valvular disease

SPECIAL DIFFICULTIES

1 SEPTICÆMIA Often identical

2 ENTERIC -In endocarditis, note rigors, leucocytosis, may be rapid onset, no agglutination, etc

3 SIMPLE ENDOCARDITIS — Severity and progress of symptoms.

4 MALARIA --Blood examination.

Prognosis.—Invariably fatal (except possibly chronic cardiac form with negative blood culture).

Treatment — Palliative Mercury and other drugs are under trial intravenously.

CHAPTER CXI.

CHRONIC VALVULAR DISEASE.

I. AORTIC INCOMPETENCE.

Results from (1) Disease of valves, or (2) Enlargement of orifice
Commoner in males, especially strong middle aged men

Causes.—

1 CONGENITAL MALFORMATIONS Rare Usually fusion of cusps Subsequently, chronic endocarditis
2 ENDOCARDITIS —In children and young adults
MORBID ANATOMY —As in *rheumatic endocarditis* vegetations sclerosis, and occasionally calcification Often some stenosis also, and mitral valve affection.
3 SYPHILITIC AORTITIS —Commonest cause in middle age Wassermann reaction positive
4 ARTERIOSCLEROSIS.—Especially in later life
MORBID ANATOMY —As in chronic endocarditis commonly valve surface smooth, no vegetations Stenosis unusual Ancillary changes (*a*) Arteriosclerosis of aortic arch, whence interference with coronary arteries, (*b*) Atheroma
5 RUPTURE OF A VALVE With healthy valves, very rare From sudden strain, not necessarily severe on valve previously diseased, especially in infective endocarditis
6. RELATIVE INCOMPETENCE From dilatation of aortic ring Uncommon Occurs in (*a*) Arteriosclerosis of aorta with dilatation above valves (valves usually arteriosclerotic also); (*b*) Aneurysm above valves Occurrence doubtful from dilatation of left ventricle alone

Note —*Stenosis* uncommon except in endocarditic group

Effect of Aortic Incompetence Blood regurgitates from aorta to left ventricle, whence: (1) Deficient blood in systemic vessels (i e, anæmia), (2) Overfilling of left ventricle
COMPENSATION —Overfilling of left ventricle causes dilatation followed by hypertrophy Hypertrophy, by increasing output, corrects anæmia, dilatation corrects overfilling Hence, *compensation established* and symptoms slight, though reserve power is diminished

OTHER CHANGES IN HEART.—
Dilatation and hypertrophy of left auricle associated with relative incompetence or disease of mitral valves.
Dilatation and hypertrophy of right heart in chronic cases
In arteriosclerotic group: stenosis of coronary arteries, whence fibroid myocarditis. (Coronary circulation probably affected also in all forms by fall of diastolic pressure.)

In endocarditic group may be 'dynamic dilatation of arch', no post mortem changes

Systemic arteries, often sclerosed by high systolic pressure

Size of heart Often 20 to 40 oz (cor bovinum)

CARDIAC FAILURE May result from (1) Failure of compensatory mechanism, or (2) Onset of abnormal cardiac rhythms Also from (3) Sudden occurrence of incompetence e g, ruptured valve

Symptoms —May be latent

1 EARLY SYMPTOMS Headache (often throbbing) giddiness faintness on rising or stooping flashes of light often irritability of temper, and defective memory Shortness of breath Palpitations on exertion Pain often severe, character varies (*a*) dull precordial ache (*b*) sharp pain radiating along arms usually left (*c*) attacks of angina, (*b*) and (*c*) depend on changes in aorta and coronary arteries (*see* ANEURYSM) Anæmia often marked

2 FAILING COMPENSATION —Shortness of breath, and discomfort Cardiac pain Œdema of feet Nocturnal attacks of dyspnœa and orthopnœa Cough œdematous or congested lungs Bad dreams disturbed and restless sleep very common Mental symptoms common towards end

Uncommon *General œdema* (unless auricular fibrillation) Hæmoptysis Cyanosis Embolii rare

3 SUDDEN DEATH —Frequent

Physical Signs (in definite stage) —

INSPECTION Precordial pulsation extensive and forcible often also in 2nd right space Apex beat in 6th or 7th space, often outwards to axilla (Pulsation often traceable from apex to large peripheral arteries Between apex beat and sternum several spaces may retract during systole of ventricle)

PALPATION —Apex beat forcible and heaving, except with dilatation, when weak and wavy *Rarely* a diastolic thrill

PERCUSSION —Increase of cardiac dullness marked, especially downwards to the left

AUSCULTATION Characteristic *Diastolic murmur at base, conducted down sternum*

SITE OF MURMUR Often audible *earliest* and best to *left* of sternum, near 4th cartilage, or at mid sternum

DIRECTION OF PROPAGATION —Down sternum, rather than towards apex

CHARACTER —*Soft*, blowing, and prolonged

AORTIC FIRST SOUND —(1) Clear, or more commonly: (2) Short systolic murmur similar to that of aortic stenosis, but not proof of presence (double aortic, 'to-and-fro' murmur)

In arteriosclerosis, murmur soft, no thrill In endocarditis, often rough, from vegetations, sometimes a thrill, may also be true stenosis

Aortic Incompetence –**Physical Signs,** *continued*

AORTIC SECOND SOUND –(1) Replaced by murmur, (2) Present with murmur, or (3) Loud, if arch dilated

AT MITRAL AREA Sounds dependent on condition of valves, affections common

First Sound – (1) Clear, in compensation, (2) Systolic murmur, often loud, from relative mitral incompetence in dilatation, or from concomitant valve disease

Second Sound. – Diastolic murmur from base may be audible

Austin Flint Murmur Presystolic or late diastolic Rumbling, limited to apex variable and transient there may be slight thrill Occurs in about half of the cases Ascribed to aortic regurgitant blood pushing anterior mitral valve in path of auricular blood entering ventricle No systolic shock or loud first sound

ARTERIES –(1) Arteriosclerosis and tortuosity common (2) Throbbing visible pulsation usually marked, e g, in carotid brachial, radial, retinal arteries, abdominal aorta may be extreme On auscultating large arteries e g, femoral a to and-fro murmur or systolic shock is audible

CAPILLARY PULSE Especially seen in finger nails, lips or line drawn across forehead Occasionally in peripheral veins Due to relaxation of peripheral vessels

PULSE * –Characteristic Corrigan's 'water hammer' pulse short forcible impulse with rapid fall best felt by grasping wrist and holding arm above head

Sphygmograph High quick ascent sharp top rapid fall small dicrotic wave Characters of pulse due to compensatory dilatation of peripheral vessels, and not to regurgitation of blood

BLOOD PRESSURE —Systolic pressure high 100 to 150 mm in arm, diastolic pressure low Systolic pressure in lower extremities 50 to 80 mm higher than in upper (Hill and Holtzmann)

AORTIC FACIES—Face long drawn tired, and anæmic, distinctive from broad appearance in mitral disease

Rupture of a Valve.—*Valve previously diseased* infective endocarditis common, also aneurysm of valve Rupture invariably is due to a strain, not necessarily severe

SYMPTOMS —*Sudden cardiac pain,* immediately followed by extreme dyspnœa, great general distress, and cyanosis or earthy pallor Patient may feel 'something give way in heart' Symptoms *most urgent at onset,* and subsequently tend to improve with rest

PHYSICAL SIGNS Sudden development of aortic diastolic murmur. Rapid dilatation of heart Pulse rapid (partly psychical)

* The 'delay' of pulse from heart to radials is not supported in fact by recent investigations.

Prognosis.—Most serious of single valve lesions Death always premature gradual or often *sudden* After recognition, life rarely exceeds 10 years (*See* PROGNOSIS IN VALVULAR LESIONS)

CARDIAC FAILURE Onset accelerated by (1) Fibroid myocarditis, associated with sclerosis of coronary and other arteries (2) Auricular fibrillation is often obstinate (3) Lesions of mitral valves

ANGINA, and also PAROXYSMAL DYSPNŒA, often lead to rapid death

II. AORTIC STENOSIS.

Etiology.—Less definite than other valvular lesions as are the symptoms Associated mainly with advanced arterial changes in old men More rarely at younger ages occasionally with rheumatic factors Syphilis, rheumatism, and causal factors may all be absent *Rare lesion* Usually incompetence also present

Morbid Anatomy.—Changes may be —

(1) Valves thick and rigid may be calcified and orifice minute Common form especially in old men

(2) Valves adherent at margin with little or no thickening Mainly in younger cases

(3) Relative stenosis Aorta greatly dilated valves and ring normal

Left ventricle hypertrophied dilatation slight

Effects of Aortic Stenosis. *Hypertrophy of left ventricle* results from greater resistance to outflow often with little or no dilatation During compensation other cardiac changes slight With cardiac failure dilatation of left and right cavities and pulmonary congestion

Arteries sclerosis less marked than with incompetence (and blood pressure lower

Outflow of blood in general less than normal although systole is increased in length

Symptoms. *Latent* if compensated often for years Symptoms indefinite, and mainly referable to other lesions i.e., aortic incompetence mitral lesions arterial changes (in old age)

1 EARLY SYMPTOMS Faintness Giddiness Symptoms marked in incompetence are less definite here viz headache dyspnœa palpitations precordial pains and angina

2 WITH DILATATION AND CARDIAC FAILURE —Dyspnœa, cough, general œdema

Physical Signs.—

INSPECTION —Precordial pulsation not extensive *Apex beat* displaced downwards and slightly outwards heaving

PALPATION —*Thrill*, maximum at aortic area, often intense

PERCUSSION Area of cardiac dullness not greatly increased

Aortic Stenosis -Physical Signs, *continued.*

AUSCULTATION.—

AORTIC AREA.—

1. *Loud rough systolic murmur*, often musical; maximum at aortic cartilage; *conducted upwards* into carotids.
2. Second sound: (a) Absent—commonly (valve sclerotic); (b) If present, short sound, from low blood-pressure; (c) Diastolic murmur, frequent, from incompetence ('double aortic murmur')

MITRAL AREA.—Aortic murmur may be audible, or murmur of mitral incompetence.

PULSE.—Regular, slow, small long-drawn wave, tension hard

Sphygmograph: Slow rise, sustained summit, slow fall. Often anacrotic. Occasionally bisferiens (double wave at summit): cause unknown.

NOTES ON PHYSICAL SIGNS.—

AORTIC SYSTOLIC MURMURS are common, and usually due to causes *other than* aortic stenosis, viz.—

1. Rough or calcified valves or vegetations, without narrowing.
2. Rough or atheromatous aorta.
3. Aneurysm or dilatation of aorta. Murmur may be marked, also thrill; but usually loud second sound is present, and other signs.
4. Hæmic murmurs in anæmia. Murmur faint, no thrill, no hypertrophy.
5. Aortic incompetence. Murmur not loud, no thrill.

In stenosis, the murmur is usually *louder*, rougher, and more musical than in other conditions.

Aortic murmurs of any origin are often audible posteriorly.

AORTIC THRILL.—Also felt in aneurysm, and occasionally *slightly* in roughened valves and aorta.

The heaving forcible apex beat associated with the small pulse contrasts with condition in incompetence.

Diagnosis.—Characteristic, especially in old men, are: (1) Aortic thrill. (2) Rough aortic systolic murmur, conducted into carotids. (3) Hypertrophy of left ventricle, with little dilatation. (4) Pulse: slow, small, hard, long wave.

No characteristic symptoms.

Aortic systolic murmurs are common *without narrowing*, and diagnosis of stenosis is justified only in presence of a thrill.

Prognosis depends considerably on condition of arteries and other valve lesions. Is regarded as least serious valvular lesion, but statistics show that life rarely exceeds a few years after diagnosis.

III. MITRAL INCOMPETENCE.

A condition in which the normal closure of the left auriculo-ventricular valves does not occur, thus permitting a reflux of blood from ventricle to auricle.

Etiology.—Two forms: (1) Valvular incompetence; (2) Muscular incompetence.

1. VALVULAR INCOMPETENCE.—Due to 'organic' changes in valves and valve ring.

   CAUSE.—Endocarditis, of rheumatic origin: exceptions rare

   MORBID ANATOMY.—Thickening, deformity, contraction, and union of valves; often changes in and shortening of chordæ tendineæ and papillary muscles. Valve ring generally thickened and contracted, may be calcified. Near ring, endocardium often thickened, and (microscopically) myocardial changes (See ENDOCARDITIS.)

   Note.—Mitral stenosis in some degree is rarely absent.

2. MUSCULAR INCOMPETENCE. Normal valves, functioning imperfectly. Occurs in:

   a. DILATATION OF LEFT VENTRICLE. As in: (i) Aortic disease; (ii) Chronic nephritis; (iii) Arteriosclerosis, (iv) Adherent pericardium; (v) Fatty heart.

   b. WEAKNESS OF CARDIAC MUSCLE. In (vi) Anæmia (vii) Fevers.

Effects of Mitral Incompetence (*see also* CARDIAC INSUFFICIENCY).

1. CHANGES IN THE HEART.

   (a) During ventricular systole, blood regurgitates. Auricle *dilates* from increased contents, and *hypertrophies* from increased work in expulsion.

   (b) Left ventricle thus receives increased flow from auricle. Hence left ventricle similarly *dilates and hypertrophies*.

   (c) Emptying of pulmonary veins is impeded by ventricular flow. Hence right ventricle *dilates and hypertrophies*.

   Later: Right auricle dilates and hypertrophies. Pulmonary arteries and veins dilate; often become atheromatous.

2. COMPENSATION. Dilatation and hypertrophy as above, proceeding simultaneously and parallel with progress of lesion, result in normal peripheral circulation, often maintained for years: thus the lesion is 'compensated', mainly by hypertrophy of the two ventricles.

   In muscular incompetence, compensation is less complete, owing to weakness of muscle.

3. CARDIAC FAILURE: DISTURBANCE OF COMPENSATION.—The balance so established may be disturbed by various factors:—

   TYPE *a*.—(i) Recurrent endocarditis: increased incompetence. (ii) Affections of the lungs. (iii) Intercurrent diseases and fevers.

   TYPE *b*. Abnormal cardiac rhythm, especially auricular fibrillation.

Symptoms.—No symptoms may occur during development of lesion and subsequently, if compensation efficient, except *shortness of breath on exertion*.

1. MINOR SYMPTOMS WHILE COMPENSATION EFFICIENT.—*Shortness of breath on exertion*: invariable. Commonly:

Mitral Incompetence—Symptoms, *continued.*

Palpitations, attacks of bronchitis (from pulmonary congestion) *Facies* Broad and ruddy, venule on cheek dilated, cyanotic tinge of lips and ears, often suggestive

2 COMPENSATION FAILING —

CHARACTERISTIC EARLY SYMPTOMS (a) *Palpitations* irregular pulse, dilatation of heart (b) *Dyspnœa* on slight effort later in severe paroxysms (c) *Cough* much sputum often *hæmoptysis*, signs of œdema or consolidation at bases (d) *Œdema of feet*

LATER General venous engorgement often icteric tint *'Sleep starts'* and restlessness *Œdema* spreads upwards to body (general anasarca), and to serous cavities especially right hydrothorax *Urine* scanty, concentrated albumin present *Liver* becomes large and tender Rarely *emboli* in brain kidney, etc

RECOVERY may occur with rest and treatment

3 FINAL STAGE —Great suffering Lividity Orthopnœa Constant restlessness and insomnia May be vomiting General œdema Abdominal discomfort from ascites and tender liver Position of maximum ease sitting in chair with trunk bent forward and arms lying on a rest

DEATH Usually after recurrent attacks of failure Sudden death rare

Physical Signs.—

INSPECTION —(1) *Visible pulsation* increased when advanced, also to right of sternum and continuous up to cervical veins (2) *Apex beat* displaced outwards often in 6th space

PALPATION *Apex beat* forcible With failure feeble and wavy

PERCUSSION —*Area of cardiac dullness* increased, especially in *width*, right and left of sternum (both ventricles enlarged)

AUSCULTATION —Characteristic are —

MITRAL AREA —Systolic murmur

Note —(1) Character of mitral systolic murmur Maximum at apex conducted into axilla, soft and blowing or loud and musical, loudest at onset and fades off gradually replaces first sound partly or completely (2) Presystolic murmur common (3) When failure occurs, soft systolic murmur is heard at tricuspid area if severe often loud systolic only at all areas

PULMONARY AREA Second sound accentuated

PULSE —In compensation full and regular practically normal Irregular with onset of symptoms, and irregularity may persist after recovery.

Estimation of Degree of Regurgitation.—Loud systolic murmur is little guide occurs with (1) Small leak (especially 'high pitched' murmur) (2) Good muscular compensation, (3) Large regurgitation Accentuated pulmonary second sound evidence of good compensation and against great regurgitation

✔ Severe incompetence suggested by: (1) Forcible apex beat with small pulse (much reflux blood); (2) Great width of cardiac dullness.

Diagnosis.—Usually simple. Characteristic: (1) Apical systolic murmur, conducted outwards; (2) Accentuated pulmonary second sound; (3) Lateral increase of cardiac dullness; (4) Frequently, rheumatic history; (5) Presystolic murmur proves organic valvular disease.

Diagnosis (*see* CARDIAC SOUNDS AND MURMURS) from: (1) Functional murmurs. (2) Relative valvular incompetence: in conditions of cardiac enlargements.

Mitral Incompetence in Children under 12 Years.—In acute rheumatic fever, over half develop valvular disease, mainly incompetence. Of cases of incompetence, about a third give no rheumatic history, but are otherwise indistinguishable: probably "endocarditis may be sole rheumatic manifestation" (Garrod).

NOTES ON MORBID ANATOMY. Pure incompetence without stenosis not uncommon (in adults very rare). *Pericarditis* frequently co-exists in rheumatic cases (in adults rare): prognosis bad.

NOTES ON SYMPTOMS. *Œdema*: Usually general, not ascending from feet. Recovery from attacks of failure usually good. *Deficient growth and nutrition* common with incomplete compensation: often deformity of thorax. Slow and delayed development during *puberty* improves prognosis.

✔IV. MITRAL STENOSIS.

Obstruction to the blood-stream resulting from changes in the left auriculo-ventricular valves and ring.

Etiology.—Two groups:—

✔1. History of acute endocarditis: rheumatic or choreic.

✔2. No record of illnesses of rheumatic type. This group was formerly considered "congenital", but congenital mitral lesions are extremely rare. Probably it also is rheumatic, and, as in incompetence, "endocarditis may be sole rheumatic manifestation" (Garrod).

RELATION TO ACUTE RHEUMATIC ATTACK.—Physical signs of stenosis never develop during attack: stenosis is the result of slow sclerosis: minimum possibly six weeks, usually months or years.

SEX.—Commoner in females, about 2 to 1 male. Ascribed to greater frequency of rheumatism and chorea.

AGE.—Symptoms become manifest at all ages; most commonly in young adult females, 20 to 30 years.

Morbid Anatomy (*see also* ENDOCARDITIS).—

CHANGES IN THE VALVES.—Adhesions, thickening, contraction, and calcification of mitral valves result in two types:—

(1) CORRIGAN'S BUTTON-HOLE CONTRACTION.—Usual form in adults. General changes of segments and ring result in flat firm mass with a slit as aperture.

Mitral Stenosis—Morbid Anatomy, *continued.*

(2) FUNNEL-SHAPED STENOSIS.—Usual form in childhood. Cone formed by adhesion of valve edges, with little thickening. Aperture may admit tip of little finger: in advanced cases only a pencil or probe.

OTHER CARDIAC CHANGES—

(1) Hypertrophy of left auricle (firm muscular walls) and right ventricle. Left ventricle small. Total increase of heart, medium: weight 12 to 16 oz.

(2) Ante-mortem thrombi in left auricle, especially appendix (whence emboli).

Effects of Mitral Stenosis.—Closely similar to mitral incompetence (q.v.), except: (1) Sequence of affected chambers; (2) Disturbances of rhythm frequent.

1. SEQUENCE OF CHAMBERS AFFECTED. (a) Left auricle hypertrophies in driving blood through stenosed orifice. (b) Right ventricle hypertrophies and dilates. Left ventricle remains small, receiving little blood.

2. DISTURBANCES OF RHYTHM. Myocardium is often affected by rheumatic process, whence frequency of *auricular fibrillation*, heart-block, and paroxysmal tachycardia.

WITH AURICULAR FIBRILLATION. Presystolic thrill and murmur disappear. Signs at mitral area are: (1) Loud first sound, may be diastolic bruit. (2) Gallop rhythm, or (3) General systolic murmur. Second sound often *inaudible*. Marked irregularity in rhythm and force.

Physical signs of stenosis return with improved condition.

FAILURE OF COMPENSATION occurs from: (1) Auricular fibrillation (usually); (2) Muscular failure.

Symptoms.—Often none for years except *slight shortness of breath*. Symptoms depend on the complications.

FAILURE OF COMPENSATION.

DYSPNŒA; COUGH; PALPITATIONS AND RAPID AND IRREGULAR HEART.—

Generally resembles mitral incompetence, but note that lungs and abdomen are specially affected; thus:—

HÆMOPTYSIS is commoner and more profuse.

ŒDEMA is rarely extreme, but ascites is more frequent.

ENLARGED TENDER LIVER with ventricular pulsation is more frequent.

PULSE.—Usually markedly irregular.

Physical Signs.—

INSPECTION.—*Pulsation*: left spaces near sternum. *Apex beat* not displaced outwards.

PALPATION.—*Presystolic thrill*: almost pathognomonic, in 4th and 5th left spaces, rough and localized. *Systolic shock*: at termination of thrill, and synchronous with apex beat. *Apex beat*: palpable in 3rd and 4th spaces, often forcible, but varies. Sometimes pulsation also palpable in 2nd space (from conus arteriosus of right ventricle).

PERCUSSION.—*Area of cardiac dullness increased mainly to right of sternum.*

AUSCULTATION (*see also* CARDIAC SOUNDS AND MURMURS).—

MITRAL AREA.—(1) *Presystolic murmur*, to right of apex, localized; rough; *crescendo*, ending sharply in loud first sound. (2) *First sound* very clear and loud. (3) Second sound reduplicated commonly. (4) Mid-diastolic murmur (diminuendo) occasionally. (5) Frequently *systolic murmur* due to incompetence.

PULMONARY AREA. *Second sound* accentuated and often reduplicated.

AORTIC AREA. Usually unaltered.

TRICUSPID AREA. May be systolic murmur from incompetence.

COMBINED AUSCULTATION AND PALPATION.—(1) Presystolic thrill and murmur are synchronous. (2) Systolic shock, loud first sound, and apex beat are synchronous (systole well timed by finger on carotid artery).

PULSE.—Small. Completely irregular when auricular fibrillation supervenes.

LUNGS. The enlarged heart frequently presses on left bronchus, producing collapse and signs of consolidation at left base.

VENOUS PULSATION. With failing compensation and auricular fibrillation, systolic regurgitation of blood occurs into: (1) Cervical veins: pulsation and enlargement. (2) Liver: enlarges and pulsates (in systole).

Complications.—

1. BRONCHITIS and pulmonary conditions. Always serious.
2. RECURRENT ATTACKS OF ENDOCARDITIS.
3. EMBOLISM. Special danger in mitral stenosis, from (a) Thrombi in left auricle, (b) Fragments detached from the valves, less frequently. Usual sites: (i) *Cerebral vessels*, usually motor area, whence paralyses and aphasia (left middle cerebral artery). (ii) Spleen: pain in left side. (iii) Kidney: lumbar pain, followed by hæmaturia.
4. DISTURBANCES OF RHYTHM.—Especially *auricular fibrillation*: occurs in majority of cases.
5. HÆMOPTYSIS.—Occasionally severe. Fatal very rarely.

Rarely:—

6. PARALYSIS OF LEFT VOCAL CORD.—Left recurrent laryngeal nerve inflamed by pressure between aortic arch and left pulmonary artery.

Diagnosis.—Simple when condition is fully developed.

CHARACTERISTIC PHYSICAL SIGNS.—(1) Presystolic thrill, ending in systolic shock; (2) Presystolic murmur, ending in loud sharp first sound; (3) Pulmonary second sound accentuated and often reduplicated.

IN EARLY STAGES, when developing, may be suggested by: (1) Presystolic murmur transiently present after exertion, or systolic murmur; (2) Second sound reduplicated at apex and not at base.

IN LATER STAGES (auricular fibrillation), may be undiagnosable.

Mitral Stenosis - Diagnosis, *continued.*

PRESYSTOLIC MURMURS ALSO OCCUR IN:

1. AORTIC INCOMPETENCE.—Austin Flint murmur. Soft murmur; other signs different. Difficulty rare.
2. ADHERENT PERICARDIUM.—Mitral stenosis not infrequently co-exists, but presystolic murmur may occur in its absence. Difficulty rare, and of little practical importance.

HÆMOPTYSIS and COUGH occasionally suggest tuberculosis Association of tuberculosis and mitral stenosis is very rare.

V. DISEASE OF THE TRICUSPID VALVE.

Rare. Usually acquired. Congenital very rare. Almost invariably other valves affected.

Tricuspid Incompetence.- Tricuspid valves become incompetent with very slight increase of pressure Two groups:

1. ORGANIC, FROM ENDOCARDITIS Very rare.
2. RELATIVE INCOMPETENCE.—Common Occurs as sequel of: (*a*) Lesions of mitral and aortic valves; (*b*) Chronic obstruction of pulmonary circulation, e.g., bronchitis and emphysema.

PHYSICAL SIGNS.- Characteristic are:-

1. SYSTOLIC PULSATION IN CERVICAL VEINS. Jugular greatly dilated. From regurgitation.
2. HEPATIC ENLARGEMENT, WITH SYSTOLIC EXPANSILE PULSATION.
3. SYSTOLIC MURMUR OVER LOWER STERNUM. Soft; localized, or less often conducted to the right. Usually distinguishable from co-existent mitral murmur.

Other signs are:—

1. Pulsation to right of sternum and in epigastrium.
2. Area of cardiac dullness increased to right.

Venous pulse tracings show that regurgitation may occur without a systolic murmur.

SYMPTOMS.—Due to venous and pulmonary congestion, and to the co-existent valvular lesions. Dyspnœa and orthopnœa marked.

Tricuspid Stenosis.—Rare. Diagnosis unusual. Of rheumatic origin (as in mitral stenosis), and not congenital. Mitral stenosis almost invariably present. Occasionally aortic disease.

PHYSICAL SIGNS.—Usually indefinite. May be:—

1. Presystolic pulsation in cervical veins (venous tracing) and in liver. The most constant sign.
2. Presystolic thrill over sternum, and systolic shock. First sound accentuated.
3. Presystolic murmur over sternum. *Rarely* present.
4. Cardiac dullness increased to right.

SYMPTOMS.—Cyanosis, dyspnœa, and œdema marked. Also enlarged liver. Symptoms of cardiac failure.

Congenital Affections of the Heart—Varieties, *continued*

4. TRANSPOSITION OF AORTA AND PULMONARY ARTERY
   - Many varieties Associated with various defects of chambers and septa Occasionally cavities normal May be no symptoms and no murmur or hypertrophy
5. PATENCY OF FŒTAL PASSAGES
   a FORAMEN OVALE See DEFECTS OF THE CARDIAC SEPTA
   b DUCTUS ARTERIOSUS Associated with stenosis at pulmonary orifice deficient ventricular septum, and other defects Normally closed before fourteenth day of life

Symptoms—

1. CYANOSIS (*morbus cæruleus*) blue babies)—Classical symptom Noticed at end of first week of life or later Degree extreme Generalized or extremities only often general duskiness of skin
   CAUSE OF CYANOSIS Much discussed Theories (1) Deficient aeration of blood in lung (2) Venous congestion Probably both are factors also *erythræmia* and excessive hæmoglobin The tint is brightish blue combining asphyxia and congestion Most constant in pulmonary stenosis Frequently absent in case of single ventricle disproving theory of mixed arterial and venous blood
2. CLUBBED FINGERS Often too late
3. DYSPNŒA May be paroxysmal tendency to cough and bronchitis
4. ERYTHRÆMIA Red cells 7,000,000 to 10,000,000 also increased percentage of hæmoglobin

Subjects small and weakly Convulsions common Surface temperature often low

Physical Signs.—Diagnosis usually easy, but determination of exact lesion difficult or impossible Common signs are (1) *Loud long systolic murmur* maximum towards base and upper third of sternum, widely transmitted (2) Area of cardiac dullness either not increased or increased to the right May be no other signs and these occur in a variety of lesions

CONGENITAL PULMONARY STENOSIS

1. *Thrill, fine, systolic,* maximum in second left space, may be widely transmitted
2. *Systolic murmur,* maximum towards base
3. *Pulmonary second sound very weak*
4. Area of cardiac dullness increased to right

Note Thrill may be absent Rarely pulmonary second sound loud probably stenosis of conus arteriosus or pulmonary stenosis with widely patent ductus arteriosus

PATENT DUCTUS ARTERIOSUS.—When large, signs distinctive—

1. *Murmur of peculiar 'rushing' character* maximum at 3rd left space, and conducted outwards, onset after clear first sound, but almost continuous, with systolic intensification
2. *Pulmonary second sound accentuated*. often reduplicated

CHAPTER CXIII.

CONGENITAL AFFECTIONS OF THE HEART.

Many of these affections are incompatible with life. Subjects who survive infancy are mainly those with lesions of the pulmonary orifice. Malformations elsewhere in body often co-exist. Right side of heart affected much more commonly than left.

Etiology.—(1) *Mal-development*; (2) *Fœtal endocarditis.* The forms often co-exist and are difficult to distinguish in valvular lesions.

FŒTAL ENDOCARDITIS.—

SITE.—Pulmonary valves most commonly.

MORBID ANATOMY.—*Sclerosis.* Valves thicken at edges, unite, and contract; very smooth; vegetations very rare. (1) Pulmonary valves (less often aortic): valves often completely united, forming ring with narrow orifice. (2) Mitral and tricuspid valves: edges fuse: chordæ tendineæ thick and shortened: rare.

PREVALENCE ON RIGHT SIDE ASCRIBED TO: (1) Higher blood-pressure; (2) Malformations commoner, with subsequent endocarditis; (3) Toxins from placenta.

DIAGNOSIS.—Not to be confused with: (1) Fibrous nodules on auriculo-ventricular valves at birth—common. (2) Small hæmatomata at valve edges, especially mitral: probably from rupture of valvular blood-vessels, shortly before or after birth. Both groups disappear in early infancy.

Varieties.—(1) General misplacement and anomalies; (2) Defects of the cardiac septa; (3) Anomalies and lesions of the valves; (4) Transposition of aorta and pulmonary artery; (5) Patency of fœtal passages.

1. GENERAL MISPLACEMENTS AND ANOMALIES.—Often associated with various monsters—e.g., acardia, ectopia cordis.

DEXTROCARDIA.—Compatible with life; usually with complete transposition of viscera; rarely partial transposition; very rarely of heart only.

2. DEFECTS OF THE CARDIAC SEPTA.—

(a) AURICULAR AND VENTRICULAR SEPTA BOTH ABSENT, partially or completely—i.e., *cor biloculare*: single vessel supplies systemic and pulmonary circulation.

(b) AURICULAR SEPTUM DEFICIENT, especially at foramen ovale.

Varieties of Defective Foramen Ovale.—

(i) Membrane incompletely attached, leaving slit: normal for two to three months: no importance. Also minute perforations.

(ii) Membrane deficient: (α) Without other defects: common: little importance. (β) Often with pulmonary stenosis and patent ventricular septum.

(2) VENTRICULAR SEPTUM DEFICIENT: (i) Completely: *cor triloculare*: with some defect of arterial trunks. (ii) Partially: especially pars membranacea—'undefended space' (the area of this is small, and deficiency almost always involves portions of the muscular septum).

Defective Pars Membranacea.—(a) Without other defects: compatible with fair life. (b) Often with other defects: pulmonary stenosis, patent ductus arteriosus.

3. ANOMALIES AND LESIONS OF THE VALVES.—Irregularities of the auriculo-ventricular valves are rare.

a. NUMERICAL IRREGULARITIES.—

i. *Bicuspid Semilunar Valves.*—Not very rare. Especially aortic valve. One valve normal and two united. Important, since combined valve is thickened, becomes sclerosed, and aortic regurgitation results and is fatal. Etiology doubtful, whether mal-development or endocarditis.

ii. *Supernumerary Valves.*—Commonest is small fourth valve at pulmonary orifice. Little importance.

b. LESIONS AT PULMONARY ORIFICE. Commonest congenital lesions to survive childhood, especially stenosis.

(1) *'Congenital Pulmonary Stenosis', Stenosis of the Valves.*—Most important congenital lesion. Sclerotic endocarditis causes fusion of valves and advanced stenosis. Usually very smooth. May be developmental factor also. Compatible with life to greater extent than other serious defects (death from phthisis common).

Associated defects: Patent ventricular septum, sometimes patent foramen ovale, pulmonary artery usually small and aorta dilated; ventricular septum may be displaced to left and aorta communicate with both ventricles. Ductus arteriosus usually closed.

ii. *Atresia or Obliteration of Trunk of Pulmonary Artery.*—Rarer than above. Artery contracted or obliterated (forming fibrous cord) for varying length. Due to irregular division of common arterial trunk. Usually other defects present. Essential for life are patent ductus arteriosus and either patent ventricular septum or patent foramen ovale. Right heart is hypertrophied.

iii. *Stenosis of Conus Arteriosus.*—Not infrequent with stenosis at orifice. Often with patent ductus arteriosus, foramen ovale, or ventricular septum.

c. LESIONS AT AORTIC ORIFICE.—Rare. Stenosis and atresia occur. (*See above*, NUMERICAL IRREGULARITIES.)

Coarctation of the Aorta.—Rare. Narrowing at site of obliterated ductus arteriosus, with dilatation both above and below, and then usually narrowing again. Complete occlusion very rare. Anastomosis through arteries about scapula. Often other malformations.

Less constant :—

3. Thrill : systolic or almost continuous ; maximum in 2nd left space ; if conducted towards clavicle (along pulmonary artery), is pathognomonic.
4. Dull area to left of sternum over dilated pulmonary artery (Gerhardt's ribbon-shaped area).
5. Pulsation in 2nd left space, and palpable shock of closing pulmonary valves.

The above signs, when present, are diagnostic.

DEFECTS OF SEPTA.—Not distinguishable.

TRANSPOSITION OF ARTERIAL TRUNKS.—May be no abnormal signs.

COARCTATION OF AORTA.—

1. Arteries about scapulæ greatly dilated and pulsating : anastomosing arch and descending aorta : also arteries of head, neck, and arms, and possibly femorals.
2. Pulsation absent in abdominal aorta.
3. General cardiac hypertrophy.
4. Systolic murmur widely audible : other murmurs variable.

Diagnosis.—Occur in children or from childhood. Presence of : (1) Cyanosis (2) Systolic murmur : maximum intensity and conduction not corresponding to acquired murmurs. (3) Erythræmia. Usually, (4) Hypertrophy of right heart.

Duration.—Congenital pulmonary stenosis passes twelfth year oftener than any other serious defect ; rarely exceeds 25 years.

Pure defect of ventricular septum, patency of ductus arteriosus, coarctation of aorta, some transposition of vessels, and various minor defects, may pass middle age.

Death from : failure of heart ; lung complaints. Occasionally : convulsions. Rarely : cerebral abscess, infective endocarditis. In adults : tuberculosis.

Treatment.—Hygienic. Protect against colds and over-exertion. Venesection for severe dyspnœa. For cardiac failure, as in acquired conditions.

CHAPTER CXIV.

ANGINA PECTORIS.

Paroxysmal cardiac pain ; in its severer form associated closely with disease of the coronary arteries.

Clinical Groups.—The pain and symptoms may be of every grade of severity in either form, yet in general two groups can be recognized, differing etiologically, pathologically, and clinically, especially in the symptoms less locally cardiac. Characteristic forms of these two groups are :—

(1) SEVERE ANGINA (*true angina, angina major*).—Middle-aged or elderly males. Sclerosis of coronary arteries ; often syphilitic history. Pain of great severity. Sudden death not uncommon.

Angina Pectoris—Clinical Groups, *continued.*

(2) MILDER FORM (*pseudo-angina, vasomotor angina, angina minor*).—Usually females. Neurotic or vasomotor phenomena. No arteriosclerosis. No sudden death.

In addition to above there may be recognized :—

(3) MILD FORMS.—With precordial discomfort.

1. **True Angina.**—

ETIOLOGY.—

AGE.—Rare under 30 years : usually later decades.

SEX.—Rare in females.

OCCUPATION.—Mainly professional classes, acquainted with strain and worry.

PREDISPOSING FACTORS.—(1) *Sclerosis of coronary arteries* ; (2) Syphilis, especially in cases under 40 years ; (3) Arteriosclerosis of any origin.

EXCITING CAUSES.—(1) Exertion ; (2) Emotion ; (3) Flatulence ; (4) Chill.

MORBID ANATOMY.—Causal conditions are : –

(a) SCLEROSIS OF CORONARY ARTERIES —Characteristically *obliterative endarteritis* : predominant lesion. Orifices alone may be narrowed. Occasionally embolus, thrombus, or calcification.

(b) SYPHILIS.—*Aortitis* above valves ; aneurysm.

Cardiac conditions commonly associated are ancillary to : (i) *Fibroid myocarditis* ; (ii) *Aortic incompetence* ; (iii) *Syphilitic aortitis* ; (iv) *Aortic aneurysm.*

Cases are recorded without apparent lesion.

THEORY OF ORIGIN OF ATTACK.—

ALLAN BURNS' THEORY (most probable)—Coronary arteries admit sufficient blood for simple needs of heart, but insufficient for increased calls ; hence exhaustion of heart muscle.

Theory supported by : –

(a) 'Intermittent claudication' in limbs ; similar phenomena of pain and rapid exhaustion on exertion ; associated with arteriosclerosis.

(b) Exciting stimuli are such as produce vasomotor constriction of peripheral vessels or increased blood-pressure : hence increased work for heart.

(c) Vasomotor phenomena marked in pseudo-angina.

(d) Other signs of cardiac exhaustion often present—e.g., cardiac asthma, pulsus alternans.

Note.—Heart during exertion may use three times the blood of a resting heart (Starling).

SPASM OF THE CORONARY ARTERIES also suggested, producing similar results.

The condition of the heart during attack is unknown : physical signs often little changed : possibly similar to 'cramp'.

The sense of constriction is probably spasm of the intercostal muscles : a protective spasm over an injured organ (Mackenzie) ; pain similarly a protective mechanism.

SYMPTOMS.—*Exciting stimulus* invariably present. Onset usually during stimulus, occasionally delayed. Stimulus in same person often constant—e.g., exertion.

ONSET.—Sudden; rapidly attains maximum.

CHARACTERISTIC SYNDROME.—(a) *Severe pain over heart*: often radiating in definite area (*see* DISTRIBUTION OF PAIN). (b) *Sense of constriction*: as if heart compressed in vice. (c) *Mental anxiety, fear of death*: angor animi.

OTHER IMPORTANT SYMPTOMS.—(d) Subject *never walks about*; always stops if walking. 'Waves aside' proffered assistance. Usually bends, with hand pressed over heart; motionless, or changing at intervals from one position to another. (e) Vasomotor phenomena: *face ashy gray*; cold sweat. (f) *Breathing* constrained by pain.

VARIOUS AND UNUSUAL SYMPTOMS.—*Fainting* uncommon; very rarely, transient paresis and aphasia. *Pulmonary symptoms*: Paroxysmal dyspnœa ('cardiac asthma') may occur; rarely, acute pulmonary œdema (very fatal). *Abdominal angina*: Occasionally pain entirely abdominal; diagnosis difficult.

PHYSICAL SIGNS—Slight.

PULSE.—Variable; generally small and hard; may be irregular; rapidity unusual.

HEART-SOUNDS faint.

BLOOD-PRESSURE high.

DISTRIBUTION OF PAIN.—Often characteristic.

COMMON AREA AFFECTED.—*Precordial region, left axilla, inner surface of left arm, two inner fingers.* Pain commonly starts over heart and radiates thence, rarely commences elsewhere and spreads to precordium.

WIDER AREA NOT UNCOMMONLY AFFECTED.—Neck, left shoulder, left jaw; occasionally right shoulder; but right arm is rare.

TENDERNESS.—Hyperæsthesia common in area of pain. May be numb feeling.

AREAS OF CORD AFFECTED. Dorsal 1–4; also cervical 7–8; may involve dorsal 5–9.

Note.—Between attacks, and also in other cardiac conditions, tenderness often found on lightly pinching left sternomastoid, trapezius, pectoralis major (Mackenzie).

DURATION OF ATTACK.—A few seconds to one to two minutes.

TERMINATIONS.—

a. SUDDEN OR RAPID CESSATION.—Common ending. Often passage of flatus or urine. Patient usually exhausted for several days, but sometimes no such after-effects.

b. RECURRENCES.—Attacks may follow rapidly for one to two hours. Rapid cardiac failure.

c. SUDDEN DEATH.—Always feared and possible. May occur in first attack, or after many years of recurrence.

RECURRENCE OF ATTACKS.—May recur for many years. In younger syphilitic cases, cessation has occurred.

True Angina Pectoris, *continued.*

MILD ATTACKS.—Substernal oppression or milder pains may occur. In persons subject thereto, such attacks, occurring during exertion, may act as warning, and immediate rest may abort severe attack.

2. **Pseudo-Angina** (*Vasomotor Angina*).—Subjects usually exhibit marked vasomotor phenomena: blue extremities, cold clammy hands. Onset of attack probably connected with vasomotor constriction of peripheral vessels and increased intraventricular pressure.

PAIN, *in type, severity, and distribution,* may resemble severest angina major, but more often *less extreme.* Types are differentiated rather by extracardial symptoms.

GENERAL CHARACTERISTICS (cf. TRUE ANGINA).—

FEMALES commoner.

NEUROTIC and hysterical factor present. Often weak family history. Occasionally: excessive tobacco; acute infections, especially influenza.

NO ORGANIC LESIONS. Blood-pressure often high.

EXCITING STIMULI less definite: may be faintness and feeling of cold preceding pain.

ONSET less abrupt.

WALKS ABOUT RESTLESSLY DURING ATTACK. GREAT MENTAL EXCITEMENT.

NEVER FATAL.

DIAGNOSIS from severe angina is usually possible on the above points.

3. **Mild Forms.**—Precordial discomfort or distress, of varying degrees, on exertion or emotion. Regarded, correctly, by subject as sign that such stimuli have been excessive.

Prognosis.—

1. SEVERE ANGINA.—

a. IMMEDIATE PROGNOSIS IN ATTACK.—Occurrence of sudden death is rare compared with number of attacks, but may occur at any time.

b. REMOTE PROGNOSIS.—(i) Syphilitic cases under 40 years: treatment may effect great improvement. (ii) Attacks following exertion, with advanced arteriosclerosis and high blood-pressure: prognosis most serious. (iii) Following emotion: less serious.

2. PSEUDO-ANGINA.—Prognosis good. Attacks often cease.

Treatment.—

1. SEVERE ANGINA.—

a. DURING ATTACK.—Amyl nitrite inhaled: capsule 3 to 5 minims: may need several. If flatulence, hot peppermint-water and carminatives. If recurrent, inject morphia gr. $\frac{1}{4}$ and atropine gr. $\frac{1}{100}$. In great severity, chloroform anæsthesia. With signs of cardiac and respiratory failure, give

stimulants, brandy or spiritus ætheris ʒj, with spiritus ammoniæ aromaticus ʒj, in a little water. If cyanosis marked, oxygen.

b. BETWEEN ATTACKS.—Of highest importance. As in compensated cardiac conditions (*see* p. 697). If blood-pressure high, pot. iodide gr. x and liq. trinitrini ♏j, t.d.s. Treat flatulence and dyspepsia especially.

SYPHILITIC CASES.—Antisyphilitic remedies.

Note.—Wassermann reaction should always be tested.

2. PSEUDO-ANGINA. Treat general condition. If recurrent, Weir-Mitchell treatment.

CHAPTER CXV.

ARTERIOSCLEROSIS.

Thickening and degeneration of the arterial coats, local or general. Includes various pathological and clinical types.

Etiology.—Difficulty results from undoubted fact that several factors may be either the cause or result of arteriosclerosis. Consider the three closely associated conditions, high blood-pressure, arteriosclerosis, and chronic interstitial nephritis. Arteriosclerosis, of any origin, usually results in high blood-pressure and chronic nephritis: conversely, it may result from high pressure or chronic nephritis. Further, different factors produce different types of arteriosclerosis. Influence of internal secretions, e.g., suprarenal, is at present uncertain. Main factors resulting in arteriosclerosis are:—

1. HIGH BLOOD-PRESSURE. Chronic hypertension may be:—

PRIMARY (Allbutt's '*hypertonia*').—No previous renal, cardiac, or arterial disease, but is a cause of arteriosclerosis. May arise from: (i) General strain: hard workers and hard livers; over-eating. (ii) Prolonged over-exertion of muscles.

(*Pathological types*: Diffuse arteriosclerosis; also atheroma.)

SECONDARY to chronic nephritis, arteriosclerosis, lead.

Influence of high pressure on arteriosclerosis proved by: (i). Rare occurrence in pulmonary arteries; (ii) Occurs in pulmonary arteries when pressure is high, e.g., in mitral stenosis; (iii) Frequency at points of strain, viz., arch of aorta, orifice of branches.

Note.—Normal arterial systolic pressure in mm. of Hg is approximately the age in years plus 100, but there are wide variations consistent with perfect health.

2. SENILE INVOLUTION CHANGES.—In old age almost invariable. Sometimes at younger ages: frequently an hereditary or familial condition.

(*Pathological type*: Senile arteriosclerosis—viz., Mönckeberg's medial degeneration; also is factor in atheroma.)

3. CHRONIC INTERSTITIAL NEPHRITIS.—Two groups:—

a. Nephritis primary; arteriosclerosis and high blood-pressure secondary.

Arteriosclerosis—Etiology, *continued.*

(b) Arteriosclerosis and high blood-pressure primary; nephritis secondary.

(*Pathological types*: Atheroma, diffuse arteriosclerosis.)

4. CHRONIC INTOXICATIONS.—Especially *lead, tobacco,* and *gout.* Rarely acute infections (typhoid). Alcohol doubtful as single factor.

(*Pathological types*: Atheroma, diffuse arteriosclerosis.)

5. SYPHILIS.—Action on arteries of highest importance, but causes neither atheroma nor diffuse arteriosclerosis.

(*Pathological types*: Mesaortitis and aneurysm, periarteritis, and obliterative endarteritis.)

Morbid Anatomy.*—The main types are: (1) Nodular, or atheroma; (2) Diffuse; (3) Senile; (4) Syphilitic (considered here for convenience). The types may be co-existent.

1. NODULAR ARTERIOSCLEROSIS (*Atheroma*).—

VESSELS AFFECTED.—Aorta and main branches.

FACTORS.—High blood-pressure, liability increasing with age. Syphilis is not a factor.

HISTOLOGY.—Earliest change, hypertrophy of intima. Then fatty degeneration in deeper layers of intima and also media, and *impregnation with lime salts.*

MACROSCOPIC APPEARANCE.—Early: slightly raised yellow patches. Later: firm 'plaques'.

SUBSEQUENT CHANGES.—(a) Mass *softens,* forming *atheromatous abscess*; (b) Abscess ruptures in lumen, forming *atheromatous ulcer.*

SEQUELÆ may be: (a) Thrombus forms on surface, occluding narrowed artery; (b) Dissecting aneurysm.

2. DIFFUSE ARTERIOSCLEROSIS ('arterio-capillary fibrosis' of Gull and Sutton, or 'diffuse hyperplastic sclerosis').—

VESSELS AFFECTED.—Larger vessels tend to escape. Primarily affects arterioles and smaller arteries, especially in kidney, spleen, and brain.

FACTORS.—Essentially the type occurring with high blood-pressure, cardiac hypertrophy, and chronic interstitial nephritis. *Age*: rare under 35 years and in extreme old age. Syphilis is not a factor.

HISTOLOGY.—Vessels thickened and tortuous: lumen diminished. *Intima*: thickening and hyaline degeneration (earliest change); later fatty degeneration. *Internal elastic lamina*: thickened; often splitting of several laminæ. *Media*: hypertrophy. *Adventitia*: fibrous tissue increased.

3. SENILE ARTERIOSCLEROSIS (Mönckeberg's medial degeneration).—

VESSELS AFFECTED.—General distribution; radial, tibial, and femoral arteries most marked; *resemble clay pipe-stems.* Large arteries also hard and tortuous.

* *See* Turnbull, *Quarterly Journal of Medicine,* 1914; and Evans, *Ibid.,* 1921.

FACTORS.—Essentially a senile change. Blood-pressure about normal and cardiac hypertrophy not associated. *Age*: rare under 50 years; increases with age. Syphilis is not a factor.

HISTOLOGY.—Fatty degeneration of media, and, later, calcification are characteristic changes; intima little affected. In aorta, calcareous plaques or slighter diffuse calcareous areas. Atheroma usually co-exists.

4. SYPHILITIC DISEASES OF ARTERIES. — Syphilis is not a cause of atheroma, and only of localized arteriosclerosis. The morbid anatomy is dealt with here for convenience.

a. AORTA.—A *mesaortitis*: essential changes are in *media*. Commonest lesion of acquired syphilis found at autopsy: is proof of syphilis. Also occurs rarely in congenital syphilis. *Spirochæta pallida* has been found (Levaditi's method). Wassermann reaction always positive.

Macroscopic Changes.—Localized, usually near root of aorta. Numerous depressed short linear or stellate scars. Rest of aorta generally normal.

Histology.—Early: (i) Perivascular infiltration of vasa vasorum extending into media; (ii) In affected areas of media, infiltration with plasma cells, degeneration of muscle cells. Later: Fibrosis of areas in media; subsequent contraction produces the linear scars.

Intima over areas may degenerate and calcify ('syphilitic atheroma'). Localized gummata are extremely rare.

Sequelæ.—Aortic valvular lesions. Aortic aneurysms.

b. SMALLER ARTERIES.—Two types:—

i. *Obliterative Endarteritis.*—Proliferative thickening of intima; elastic lamella remains unchanged and easily recognized. Later, granulation tissue forms and fibroses. Thickening may entirely fill lumen. *Similar changes occur in tuberculosis.* Giant cells occur in both forms: commoner with tubercle.

ii. *Periarteritis.*—Particularly in arterioles of brain and cord. *Adventitia* thickened and infiltrated; later, may become hyaline and fibrotic. Thickening of intima may co-exist. *Spirochæta pallida* has been found. Condition *specific of syphilis*, and marked in chronic syphilitic lesions of central nervous system.

Symptoms.—Characteristic syndrome: Middle-aged man complains of giddiness, and slight impairment of memory; arteries thickened, blood-pressure high, left ventricle hypertrophied, aortic second sound accentuated, mitral first sound roughened. Symptoms are varied, and depend on system mainly affected:—

1. CARDIAC.—*Sclerosis of the coronary arteries*, its sequelæ, and *ancillary conditions*, provide symptoms, viz: (*a*) Fibroid myocarditis; (*b*) Abnormal rhythms; (*c*) Cardiac failure; (*d*) Angina; (*e*) Coronary thrombosis and sudden death. Valvular lesions, especially *aortic incompetence*, may result from arteriosclerosis.

Arteriosclerosis—Symptoms, *continued.*

2. CEREBRAL.—(*a*) *Vertigo*—most frequent symptom; headache. (*b*) Progressive dementias; all grades from *deficient memory* to dementia. (*c*) Cerebral hæmorrhage. (*d*) Transient pareses and aphasia; ascribed to transient spasm in narrowed arteries.
3. RENAL.—Symptoms of chronic interstitial nephritis. Two types of kidney occur: (*a*) Small granular kidney (primary—arteriosclerosis secondary): urine increased in amount, specific gravity low. (*b*) Arteriosclerotic kidney - normal size, red, and firm: urine normal in amount and specific gravity.

VARIOUS SYMPTOMS. -

INTERMITTENT CLAUDICATION.—No symptoms when patient is at rest: exertion is followed by pain and tingling in legs, and, when severer, by cramp, weakness, and paresis. Ascribed to ischæmia, blood-supply being insufficient for increased work (cf. ANGINA). Vasomotor changes common: coldness and congestion; dorsal arteries of feet may be pulseless. Special factors: tobacco, syphilis.

GANGRENE OF EXTREMITIES.—From endarteritis obliterans or thrombosis.

ABDOMINAL ARTERIOSCLEROSIS.—Sclerosis of splanchnic vessels and subsequent spasm has been given as cause of lead colic and tabetic crises: possibly also cause of 'abdominal angina'.

Diagnosis.—Note: (1) Thickened arteries (examine brachial in addition to smaller arteries); (2) High blood-pressure; (3) Hypertrophy of left ventricle; (4) Condition of urine.

Treatment.—General hygienic life (*see* COMPENSATED CARDIAC LESIONS. p. 697). With high blood-pressure, pot. iod (gr v–x) and liq. trinitrini (♏ss–j) t.d.s. may relieve symptoms. In cardiac failure, treat on usual lines: early venesection indicated.

Sclerosis of Pulmonary Artery.—Occurs when pulmonary pressure is increased—e.g., in mitral stenosis, emphysema. (Sir Leonard Rogers describes a primary form in India, not uncommon, almost certainly syphilitic, and accompanied by rapid œdema.)

Sclerosis of Veins (*Phlebosclerosis*).—Not uncommon in arteriosclerosis and with high blood-pressure. Occurs in pulmonary veins in mitral stenosis. Intima thickened.

CHAPTER CXVI.

ANEURYSM.

A tumour containing blood or blood-clot and communicating directly with an artery or with the heart.

Classification.—

1. TRUE ANEURYSM (wall formed by one or more coats of the artery):—

DILATATION OR FUSIFORM ANEURYSM.—Entire circumference involved.

DISSECTING.—Blood extends between the coats. Rare.
SACCULAR.—Common type. A cavity arising from portion of the circumference, with an aperture commonly smaller than greatest diameter of cavity.
CIRSOID.—Dilatation of entire artery and its branches. Limited to small vessels.

2. FALSE ANEURYSM (communicates with an artery, but wall not formed by arterial coats).—Hæmatoma from wound or rupture of artery.
3. ARTERIOVENOUS ANEURYSM.—Direct communication between an artery and vein.
4. VARIOUS.—Parasitic; 'traction' aneurysm.

Etiology of True Aneurysms.—

AGE.—Especially 30 to 45 years. A small group in elderly men.
SEX.—Males 5 or 10 to female 1.
PREDISPOSING CAUSES—
1. SYPHILIS.—Syphilitic mesaortitis is predominant lesion and origin of aneurysms. Almost invariably present. Wassermann reaction always positive.
2. ARTERIOSCLEROSIS.—A small group occurs in elderly men, upwards of 50 years, without syphilitic mesaortitis (usually negative Wassermann reaction) but with marked atheroma.

STRAIN. Influence uncertain. Aneurysms have followed chest blows. May be exciting cause of rupture of coats when above factors are present; of no avail in their absence.
RARE CAUSES.—Infective emboli, usually multiple, in infective endocarditis. Tuberculous focus in wall of artery (very rare). 'Traction' aneurysm arising at ductus arteriosus (very rare).

NUMBER.—Usually single; rarely two or more.
SITE (in order of frequency). (1) Thoracic aorta: especially ascending and transverse portions of arch. (2) Popliteal artery: of surgical importance only. (3) Abdominal aorta and iliac arteries. Other vessels rare.
DEVELOPMENT OF ANEURYSM.—Intima atrophies or yields over area of mesaortitis, or rarely of arteriosclerosis. Pressure of blood (often high) extends dilatation. Growth of sac is opposed by: (1) Remaining tissues of wall; (2) Neighbouring structures; (3) Formation of thrombi. Sac by pressure destroys all resisting tissues, especially bone; intervertebral discs often remain after destruction of vertebræ (possibly from avascular nature).

FORMATION OF THROMBI IN SAC.—*White* thrombi deposited in successive layers; may be numerous. Harden with time; may be partial calcification; never organization.

ANEURYSM OF THE THORACIC AORTA.

DILATATION OR FUSIFORM ANEURYSM.

Site.—Ascending arch; occasionally entire arch.

Etiology.—Most common in elderly arteriosclerotic group.

Dilatation or Fusiform Aneurysm, *continued.*

Symptoms.—(1) Latent—not uncommon; (2) Co-existent aortic incompetence (either from stretching or simultaneous disease of valves); (3) Angina pectoris. Erosion of bone and pressure effects unusual.

Physical Signs.—(1) *Pulsation* in suprasternal notch, rarely in 1st and 2nd right spaces; (2) *Dullness* over and to right of manubrium; (3) *Loud aortic second sound*, or diastolic murmur, conducted upwards; (4) *X rays.*

Diagnosis.—Often not made, especially in absence of X rays.

DISSECTING ANEURYSM.

Very rare. Intima splits at weak spot, from syphilitic mesaortitis or arteriosclerosis, and blood spreads between coats. Extent variable: may form double tube for length of thoracic aorta. Split usually in ascending aorta.

Symptoms.—With large split, may be sharp pain at onset.

SEQUELÆ.—(1) Complete rupture of aorta and sudden death, inevitable if blood reaches adventitia. (2) No symptoms. adventitia and remains of media resist blood-pressure; forms 'healed dissecting aneurysm', occasionally lined by intima.

Duration may be years.

Other Allied Conditions.—

RUPTURE OF THE AORTA.—Usually complete transverse. A cause of sudden death. Etiology: syphilitic or arteriosclerotic.

RUPTURE OF INTIMA ALONE.—May heal completely.

SACCULAR ANEURYSM.

The most common type. The aorta is in propinquity to many important structures, and changes its relations rapidly, aneurysms thus producing a complexity of serious symptoms and signs. Aneurysms at the various sites are briefly considered first. A summary of physical signs and symptoms follows.

Consideration according to Site.—(1) Sinuses of Valsalva; (2) Ascending, (3) Transverse, and (4) Descending portions of the arch; (5) Descending thoracic aorta.

1. ANEURYSM OF THE SINUSES OF VALSALVA.—Not uncommon. Usually in young syphilitics. Is a cause of sudden death, by perforation into pericardium, rarely into auricle or superior vena cava.

   SYMPTOMS.—(*a*) *Latent*; (*b*) Angina; (*c*) Those of co-existent aortic incompetence.

   PHYSICAL SIGNS.—None localizing (very rarely presses on inferior vena cava, with congestion and œdema below diaphragm).

2. ANEURYSM OF THE ASCENDING ARCH.—

   Anatomy.—Aorta arises at lower border of 3rd costal cartilage, slightly to left of mid-line, and ascending arch terminates at

upper border of 2nd right costal cartilage close to sternum here separated from chest wall only by superior mediastinum, and partly overlapped by right lung and pleura. Length about 2½ inches.

ORIGIN, AND DIRECTION OF EXTENSION.—Commonly arises from convexity, and extends forwards, eroding ribs and forming external tumour in 2nd and 3rd right space. Less often extends outwards on to lungs. Very rarely arises from concavity, extending to left of sternum.

PREDOMINANT FEATURE.—*Physical signs, directly due to the tumour.* Owing to anatomical relations, both symptoms and pressure signs are usually slight.

SYMPTOMS.—(a) Pain: from slight to angina (if origin near valves). (b) Cough: paroxysms rarely. May be slight hæmoptysis.

PHYSICAL SIGNS.—Characteristic are: (a) Expansile pulsating tumour to right of sternum. (b) Accentuated aortic second sound, or diastolic murmur—i.e., *normal* second sound is strong evidence against aneurysm in this position. (c) Diastolic shock and sound over sac (important but not very common). (d) Systolic *thrill* and murmur over sac. Less definite. (e) Dull area (in absence of tumour). (f) Heart may be dislocated down to left. Unusual: Dilated veins and œdema from pressure on deep veins; inequality of pupils and pulse. Rare: Pressure on recurrent laryngeal nerve.

DEATH.—Usually from cardiac or intercurrent disease. About one-third rupture, generally into right pleura, rarely external into pericardium or superior vena cava.

ANEURYSM OF THE TRANSVERSE ARCH.—

Anatomy.—Transverse arch commences at upper border of 2nd right costal cartilage, arches across 3rd dorsal vertebra, with apex 1½ inches below sternal notch, and passing to the left and backwards, ends at left of upper border of 4th dorsal vertebra. *Crosses* bifurcation of trachea, œsophagus, and thoracic duct; *is crossed by* left recurrent laryngeal, vagus, and phrenic nerves; *below* are left bronchus and pulmonary artery.

ORIGIN, AND DIRECTION OF EXTENSION.—Usually from posterior wall, extending backwards. Less often forwards to right of sternum, from anterior wall. Rarely downwards from concavity. Sac may include innominate or carotid artery.

PREDOMINANT FEATURE.—*Symptoms, due to pressure* (often severe even if sac small).

SYMPTOMS.—(a) Alterations of voice. (b) Cough, 'bovine' or 'brassy'; often paroxysms. (c) Dyspnœa; often paroxysms. Occasionally: (d) Hæmoptysis. (e) Dysphagia.

PHYSICAL SIGNS (often slight).—(a) Suprasternal pulsation. (b) Laryngeal paralysis. (c) Inequality of pupils. (d) Inequality of pulses. (e) Tracheal tugging. Occasionally: Dilated veins and œdema from pressure on veins; dullness over manubrium. With aneurysms extending downwards

Aneurysm of Aorta—Transverse Arch, *continued.*

and pressure on bronchus: Bronchitis; bronchiectasis; collapse of lung, etc.

DEATH.—(a) Rupture, into trachea, pleura, etc. (b) Tracheal compression.

4. ANEURYSM OF THE DESCENDING ARCH.—Rarer than previous sites.

Anatomy.—Descending arch commences at upper border of 4th and ends at lower border of 5th dorsal vertebra. *In front,* root of left lung; *to right,* œsophagus; *to left,* left lung and pleura; *behind and to right,* bodies of 4th and 5th vertebræ.

DIRECTION OF EXTENSION.—Mainly backwards and to left.

PREDOMINANT FEATURES.—Arise from: (*a*) Pressure on lung structures; (*b*) Erosion of vertebræ; (*c*) Erosion of ribs posteriorly.

SYMPTOMS.—Often latent until rupture occurs. (*a*) *Pain* often referred to abdomen; severe after erosion of vertebræ and pressure on roots. (*b*) Cough. (*c*) Dysphagia. Occasionally: (*d*) Transverse myelitis.

PHYSICAL SIGNS.—(*a*) Posteriorly, in left interscapular space, pulsating tumour or dullness; systolic murmur. (*b*) Bronchitis, bronchiectasis, collapse, etc., from pressure on bronchus. (*c*) Transverse myelitis; increased knee-jerks Babinski sign, etc.

DEATH.—Rupture into pleura common.

5. ANEURYSM OF THE DESCENDING THORACIC AORTA. Uncommon.

Anatomy.—From lower border of 5th dorsal vertebra to aortic opening in diaphragm over 12th dorsal vertebra, almost in mid-line. *Is crossed* right to left by œsophagus, separating it from heart and pericardium.

ORIGIN, AND DIRECTION OF EXTENSION.—Usually close to diaphragm. Erosion of vertebræ common. Sac often very large.

SYMPTOMS.—Often latent. (*a*) Pain; often referred to abdomen; severe after erosion of vertebræ and pressure on roots. (*b*) Cough. (*c*) Dysphagia. Occasionally: (*d*) Transverse myelitis.

PHYSICAL SIGNS.—(*a*) Pulmonary. (*b*) Œsophageal obstruction. Rarely: (*c*) Pulse absent below sac. (*d*) Transverse myelitis.

DEATH.—Rupture common into pleura, also into lung; occasionally into pericardium, œsophagus, etc.

Physical Signs.—(1) Signs directly connected with the aneurysm; (2) Signs due to pressure of the sac on other structures.

I. SIGNS DIRECTLY CONNECTED WITH THE ANEURYSM. Vary with position, thickness of wall, amount of clot, and condition of heart. Especially marked in aneurysms of ascending arch.

INSPECTION AND PALPATION.—(*a*) At site of aneurysm: (i) *Pulsation*: characteristic if *expansile.* (ii) *Diastolic shock*

(ascending arch). (iii) *Systolic thrill.* (*b*) *Position of apex beat.* Usually normal, but displacement occurs from: (i) Heart dislocated: only by sacs from ascending arch. (ii) Hypertrophy: not common: results from co-existent (*α*) aortic incompetence, or (*β*) arteriosclerosis.

PERCUSSION.—*Dullness* over sac: anteriorly, or posteriorly to left of spine. May be present without pulsation.

AUSCULTATION.—(*a*) Over aneurysm: (i) *Loud second sound*, or diastolic murmur; (ii) 'Systolic' murmur. (*b*) At aortic area: Loud second sound or diastolic murmur.

X RAYS.—Frequently diagnostic: expansile tumour in line of aorta.

Notes on above Physical Signs.—

Pulsation.—Position: Near main line of aorta; anteriorly, to right of sternum or in suprasternal notch; posteriorly, to left of spine (almost always aneurysm, adherent pericardium producing 'tug' only) Pulsation anteriorly may be: (*a*) Diffuse: also in anæmia, neurasthenia, etc, and in tumours. (*b*) Expansile. If definitely *expansile* is almost pathognomonic, but clot in sac may prevent it; expansile sarcoma very rare in thorax. Large external tumour may be present.

Diastolic Shock over Sac—Not common; characteristic if present. Caused by large volume recoiling on aortic valves Hence: (*a*) Only in ascending arch; (*b*) Associated with loud second sound over sac and loud aortic second. With aortic incompetence, all three signs are replaced by diastolic murmur.

Aortic Second Sound is most important auscultatory sign: if *normal*, strongly against aneurysm of *ascending* arch.

Systolic Thrill.—Slight or intense; absent if much clot. Associated with systolic murmur. (Occurs at expansion of sac, and thus not strictly 'systolic' in time.)

Systolic murmur alone is of little importance.

2. PRESSURE SIGNS.—Especially in aneurysms of transverse arch. (*a*) Pressure on veins (*b*) Pressure on arteries: Inequalities of pulse and of blood-pressure. (*c*) Inequalities of pupils. (*d*) Pressure on air tubes and lungs: (i) Bronchitis, collapse, etc.; (ii) Tracheal tugging; (iii) Displacement of trachea (uncommon). (*e*) Pressure on recurrent laryngeal nerve.

a. PRESSURE ON VEINS—ŒDEMA AND DILATED SUPERFICIAL VEINS.—*Less common than with glandular or malignant tumours.* May be: (i) Innominate vein, left more common; (ii) Superior vena cava. Signs limited to area drained. Rarely *clubbing of fingers.* Rupture common into affected vein.

b. PRESSURE ON ARTERIES—INEQUALITIES OF PULSES.—Pulses on the two sides (*e.g.*, *radial*) may be: (i) Asynchronous; (ii) Unequal in force. Alterations due to: (*α*) Pressure of sac on arteries; (*β*) Reservoir action of sac retarding and also flattening pulse-wave.

Aneurysm of Aorta—Physical Signs, *continued.*

Typical effects.—High ascending arch: Sac presses on right subclavian artery; right radial pulse small and flat, but synchronous. Transverse arch: Pulses asynchronous, left flattened. Descending arch: Pressure on left subclavian artery; left radial pulse small and flat, but synchronous. Descending aorta: Radials normal; with large sacs, no pulse in abdominal aorta and below.

Fallacious effects arise from: (i) Stiff-walled sac with much clot; expansion slight, and pulse may be normal. (ii) Emboli: e.g., clot from ascending arch sac may lodge in left subclavian artery and diminish left pulse.

Conclusion.—Inequalities of radial pulses are common, simple, and valuable evidence of aneurysm, but uncertain as evidence of localization. They may be absent.

Blood-pressure.—There may be a difference of 20 to 30 mm. between the two brachials.

c. INEQUALITY OF PUPILS.—Causes are:—

i. Lower carotid pressure on one side, resulting in contraction of spiral arteries in iris and enlargement of pupil (Wall and Walker). *Usual cause.* Characteristics: (α) Inequality slight; often found only on shading. (β) Both pupils dilate on shading. (γ) Large pupil on side of smaller carotid pulse (if recognizable). (δ) No signs of affection of sympathetic nerve.

ii. Involvement by sac and *paralysis* of sympathetic nerve (dilator pupillæ); hence 3rd nerve unopposed. *Rare cause.* Characteristics: (α) Inequality marked. (β) Small pupil does not dilate on shading. (γ) Small pupil on side of smaller carotid pulse. (δ) Unilateral pallor, or flushing of face, sweating, and signs of sympathetic affection.

Note.—Irritation of nerve will cause dilatation, not contraction, of pupil, and phenomena will closely resemble (i).

iii. *Tabes* co-existent, with inequality of pupils. Rare.

d. PRESSURE ON AIR TUBES AND LUNGS.—

Lungs.—(i) On bronchi; produces: (α) Bronchitis; (β) Later, also dullness from collapse or retained secretion; (γ) Finally may be bronchiectasis, or suppuration. (ii) Large sac compressing lung substance: signs of collapse. Left bronchus mostly affected; hence physical signs at *left base.*

Tracheal Tugging.—Rare, except in aneurysms from transverse arch; due to pressure on bifurcation of trachea. Examine from behind, fingers under cricoid, head thrown back. Occasionally occurs in aortic incompetence, tumours.

Displacement of Trachea.—Less important. Draw line from point of thyroid cartilage to exact centre of notch.

Pressure on a Bronchus.—Suggestive of aneurysm: (i) Paroxysmal dyspnœa and cough; (ii) Physical signs at one base. Cough thus produced is not 'brassy'.

e. PRESSURE ON RECURRENT LARYNGEAL NERVE.—Most commonly left nerve, from anatomical relations; bilateral very rare. Results are: (i) Laryngoscopic: Affected vocal cord nearer mid-line, abductor and later, adductor paralysis. (ii) Alterations of voice: hoarseness, weakness; rarely complete aphonia. (iii) 'Bovine cough.

Anatomy.—Recurrent laryngeal nerve supplies all muscles of larynx except cricothyroid (tensor of cords, supplied by superior laryngeal), viz.: (i) Abductors: posterior crico-arytenoids. (ii) Adductors: remaining muscles, internal thyro-arytenoid being also tensor of cords. During life, *tone* of abductors holds cords apart.

In lesions of recurrent laryngeal, abductors always affected more and earlier than adductors; lesions are unilateral (adductor paralyses generally bilateral and functional). Three stages occur (Semon):—

Stage 1.—*Abductor paralysis*: cord assumes more central 'cadaveric position'; further adduction on phonation. Usually producing no symptoms, but found on examination. Hence *important early sign.*

Stage 2.—*Paralytic contracture of unopposed adductor muscles*, drawing cord still nearer mid-line. Symptoms: dyspnœa on exertion, with some inspiratory stridor, from narrowing of glottis.

Stage 3.—*Adductor paralysis* follows. Results: (i) Farthest muscle usually internal thyro-arytenoid, loss of tensor action causing *alteration in voice*, hoarseness, weakness. (ii) Glottis cannot be closed: hence *'bovine'* cough—viz., long noise without initial explosion. (iii) Dyspnœa increased. (iv) On examination: cord in mid-line, no movement on phonation.

VAGUS SPASM.—Rarely irritation of one vagus causes spasm of all glottic muscles, and approximation of cords by strong adductors. Hence asphyxia needing tracheotomy.

PHRENIC NERVE.—Never affected.

Symptoms.—(1) Pain. (2) Cough: (*a*) Simple; (*b*) Paroxysmal; (*c*) 'Brassy'; (*d*) 'Bovine'. (3) Dyspnœa. (4) Alterations in voice. (5) Hæmorrhage. (6) Dysphagia. (7) Rupture.

1. PAIN.—Most constant symptom; rarely absent, but very variable owing to the numerous causes; may be slight, continuous, severe, paroxysmal.

CAUSES.—

(a) *Gradual dilatation of artery* and stretching of nerve-endings—*'true aneurysmal pain'*. Reflected over certain areas, with hyperæsthesia of skin. Nerves

Aneurysm of Aorta—Symptoms, *continued.*

gradually atrophy, and hence this pain is *absent with large sacs*, and is most severe in early stages. Fairly continuous, but paroxysms also may occur: especially *nocturnal*.

b. Erosion of bones; irregular neuralgic pains.

c. Pressure on intercostal nerves; often paroxysmal.

d. Pressure on dorsal nerve roots after erosion of vertebræ; agonizing.

DISTRIBUTION OF PAIN.—When caused by:—

a. Dilatation of artery.—*Reflected from arch of aorta* over dorsal areas 1 to 4 and cervical 3, 4; also *hyperæsthesia of skin*. Approximate distribution:

Sinuses of Valsalva: Anginal pain; præcordium and left arm.

Ascending arch: On right side; from nipple to shoulder and neck, and to *back of head* (occipital headache), and sometimes inner side of right arm as low as wrist; may be frontal headache

Transverse arch: Pain more on left than right; shoulder, back of head, left arm

Descending arch and aorta: Below ductus Botalli referred to dorsal 5, 6, 7, viz, below left breast and left interscapular region; not in shoulder or arm

b. Erosion of bone—Local pain.

c. Pressure on intercostal nerves Pain *referred* to distribution of nerve; no hyperæsthesia of skin; from lower nerves radiates round *abdomen* as low as umbilicus.

d. Pressure on dorsal nerve roots Pain *referred* to distribution round *abdomen*.

CONCLUSION.—In any case of obstinate or constantly recurring pain, to account for which no cause can be found, the possibility of aneurysm should be considered.

7. COUGH.—Very rarely absent.

CAUSES.—

a. Pressure on trachea.—*Paroxysmal 'brassy'* cough; often characteristic.

b. Pressure on bronchi.—Resulting in bronchitis, retention of secretion, and occasionally bronchiectasis; often *paroxysmal*.

c Pressure on large areas of lung.—Condition may simulate phthisis.

PECULIARITIES of cough frequent.—(*a*) 'Brassy'; (*b*) 'Bovine' or 'goose' cough (recurrent laryngeal nerve); (*c*) Paroxysmal.

34. DYSPNŒA.—Constant, on exertion, or paroxysmal.

CAUSES.—Pressure on:—

a. Bronchus, especially left.—No stridor.

b. Trachea.—Inspiratory stridor; often in paroxysms. Especially in transverse arch. Frequently fatal.

c. Recurrent laryngeal nerve.—Inspiratory stridor.
d. Large areas of lung.

4. ALTERATIONS IN VOICE.—Hoarseness, weakness, rarely complete aphonia (*see* RECURRENT LARYNGEAL NERVE). May be earliest symptom.

5. HÆMORRHAGE—
   SMALL QUANTITIES.—From: (i) Exposed sac 'weeping' into trachea; (ii) Granulations in trachea; (iii) Destruction of lung alveoli. May continue for months.
   LARGE QUANTITIES. Rupture into air tubes or lungs. First bleeding often not fatal (owing to clotting), but life subsequently rarely exceeds few weeks. Rarely into œsophagus.

6. DYSPHAGIA.—*Uncommon* Most often from descending aorta. (Bougie must not be passed.)

7. RUPTURE OF SAC. Sites may be
   EXTERNAL Rupture unusual even with large external tumour. Most to right of sternum First external hæmorrhage rarely fatal owing to clotting
   TRACHEA AND BRONCHI.—Commonest site of rupture; usually left bronchus, sac erodes through.
   PLEURA.—Especially descending aorta and arch Occurs in about one third of these Rapidly fatal.
   INTO LUNG SUBSTANCE —Rarely rapidly fatal unless large air tubes opened.

Rare.—
   ŒSOPHAGUS.
   PERICARDIUM —From sinuses of Valsalva, lower descending aorta, or lower ascending arch (may dissect downwards and then rupture) Sudden death
   SUPERIOR VENA CAVA (*see* p. 724). PULMONARY ARTERY, AURICLE.

Diagnosis.—Two essential data are. (1) *Wassermann reaction* always positive in aneurysm, except rare arteriosclerotic group in elderly men. (2) *X-ray evidence.* Diagnosis from.

✓ MEDIASTINAL TUMOURS. Often difficult Note following points in tumours: (1) Sexes equal. (2) Rapid cachexia. (3) Rapid growth of tumour. (4) Secondary glands. (5) *Pulsation* not forcible; very rarely expansile; outline of tumour or dullness irregular. (6) No diastolic shock, no thrill (may be systolic murmur). (7) Pleurisy common. (8) Pressure effects less marked, *except on veins.* (9) Recurrent laryngeal nerve not involved; phrenic may be (never in aneurysm). (10) Tracheal tugging very rare. (11) Irregular pyrexia common (aneurysm is apyrexial). (12) 'Red-currant jelly' sputum in primary lung growths; otherwise scanty.

✓ DYNAMIC PULSATION OF AORTA.—May be marked in: (1) Aortic incompetence. (2) Anæmia. (3) Exophthalmic goitre, neurasthenia, and neurotic conditions.

✓ SCOLIOSIS AND DEFORMITIES, displacing heart and great vessels.—Difficulty rare.

Aneurysm of Aorta—Diagnosis, *continued*

PULSATING PLEURISY —Nowadays very rare Expansile impulse, but diffuse and not forcible. Pyrexia Leucocytosis.

Terminations.—

1. CARDIAC FAILURE AND PULMONARY AFFECTIONS.—Commonest cause, about 40 per cent
2. RUPTURE OF SAC In less than one third
3. DYSPNŒA — (Aneurysm is most frequent cause of extreme dyspnœa in adult males)
4. EXHAUSTION, SEPSIS.

Prognosis.—

GENERAL PROGNOSIS Usually one to three years after recognition Sudden death possible at any moment

SITE —Duration longest from high ascending arch Short, from transverse arch.

EFFECT OF TREATMENT (mainly rest) Relieves symptoms, and may cause reduction of sac—visibly in external tumours

SPONTANEOUS CURE (by layers of fibrin) Rare, but may prolong life five to ten years, or be discovered post mortem Only in small sacculated aneurysms

Treatment.—*Indications* (1) Reduction in number and force of heart-beats, (2) Promotion of clotting in sac all methods are unsatisfactory Treatment is mainly *palliative*.

GENERAL PRINCIPLES —

1. REST — Absolute
2. DIET — Fluids *moderately* restricted, light or milk food diet.
3. BOWELS — Regular action
4. POTASSIUM IODIDE Gr x, t d s Relieves pain action probably on syphilitic mesaortitis

Result. If maintained for weeks or months pain and symptoms are ameliorated An external sac is often visibly smaller Improvement temporary

TUFNELL'S TREATMENT — (1) Rest (2) Restricted fluid and protein diet 2 oz. fluid and 10 oz solid food: for several months Too severe, and unsupported by results

SYMPTOMATIC TREATMENT.

EXTERNAL TUMOUR —Ice bag

DYSPNŒA AND CYANOSIS —Venesection For urgent dyspnœa, tracheotomy, efficient in rare cases of bilateral abductor paralysis; if from pressure, rarely possible to get tube below obstruction.

COUGH.—Inhalations (tinct. benzoin co). Linctus heroin (gr. $\frac{1}{24}$ to $\frac{1}{12}$).

PAIN.—Ice-bag Often needs morphia.

HIGH BLOOD-PRESSURE —Nitrites

SYPHILITIC TREATMENT.—In young syphilitic patients, usual treatment. Salvarsan and similar preparations are not contra-indicated.

SPECIAL METHODS EMPLOYED TO INDUCE CLOTTING IN SAC (mentioned for completeness only)

CALCIUM LACTATE —Gr xx daily for four days, omit one week and repeat. No evidence of action

GELATIN —Subcutaneous injection, 1 per cent solution, 10 oz ; repeat every three to four days for 18 to 20 injections. *Negatived* by difficulty of sterilization and frequency of *tetanus*.

INTRODUCTION OF WIRE INTO SAC, COMBINED WIRE AND ELECTROLYSIS, SCRATCHING SAC BY NEEDLE, and similar procedures, are dangerous and valueless

VENESECTION —Repeated small amounts. **Increases** coagulability Worthy of trial

ANEURYSM OF THE ABDOMINAL AORTA.

Site of Origin —Usually close to diaphragm · cœliac axis often involved

Direction of Extension.—From anterior wall forwards into epigastrium, towards the *left* Occasionally backwards, eroding vertebræ

Symptoms.— *Pain* often intense, radiates round sides, or in back Ther may be gastric symptoms After erosion of vertebræ, compression myelitis

Physical Signs.—

INSPECTION Epigastric pulsation (rarely projecting tumour)

PALPATION *Definite tumour with expansile pulsation* (only physical sign justifying diagnosis) Occasionally movable

Often systolic murmur, may be audible at back Sometimes thrill Distal pulse usually small, may be absent

Diagnosis.— Uncommon Is frequently diagnosed erroneously Distinction by (*a*) physical signs (*especially palpation*), (*b*) X rays (*c*) Wassermann reaction, from

1. PULSATING AORTA —e g in anæmia, neurasthenia and neurotic conditions
2. TUMOURS—e g, of pancreas—lifted by aortic pulsation In these (*a*) Pulsation not forcible, (*b*) Not expansile, (*c*) In knee elbow position tumour loses pulsation.

Prognosis and Method of Termination.—Prognosis bad. Termination by :—

1 RUPTURE Usual termination Into. (*a*) Pleura, (*b*) Peritoneum, less commonly (*c*) Retroperitoneal tissues (simulating 'acute abdomen'), (*d*) Duodenum.

Occasionally —

2. EMBOLI into superior mesenteric artery ('acute intestinal obstruction'), also other branches.

Rarely —

3. COMPLETE CLOT IN SAC.
4. PARAPLEGIA.

Aneurysm of Abdominal Aorta, *continued.*

Treatment.—*See* THORACIC AORTA. *Compression* has been tried by hand, for repeated periods, under anæsthesia; some evidence o improvement; risk of damaging sac considerable.

Aneurysm of the Branches of the Abdominal Aorta.—(1) Cœliac axis: often involved in aneurysm of aorta. (2) Superior mesenteric artery: emboli may cause 'infarction' ('acute intes tinal obstruction'). Rarely: splenic artery, hepatic artery renal artery.

ARTERIOVENOUS ANEURYSM.

Two Types.—(1) *Varicose aneurysm*: 'false' aneurysm between artery and vein. (2) *Aneurysmal varix*: direct communication.

Three Characteristic Signs.—(1) *Veins distended.* (2) *Intense thrill*: maximum at site, but propagated along vessels (3) *Loud continuous murmur*, with systolic increase: similarly conducted often to a distance.

Varieties.—

1. INTERNAL ARTERIOVENOUS ANEURYSM.—From rupture of aortic aneurysms.
   - ASCENDING AORTA INTO SUPERIOR VENA CAVA.—Very rare. Symptoms: (1) At moment of rupture, sudden pain, dyspnœa, and shock; (2) Murmur as above; followed by (3) Congestion and cyanosis of upper half of body in few hours
   - ASCENDING AORTA INTO PULMONARY ARTERY.—More frequent. Symptoms as above: congestion less marked.
   - ABDOMINAL AORTA INTO INFERIOR VENA CAVA.—Very rare. Symptoms as above: congestion of lower half of body.
2. EXTERNAL ARTERIOVENOUS ANEURYSM.—Peripheral vessels. From wounds. Signs as above: varicose veins and distention of limbs often extreme, occasionally absent.

Section X.—DISEASES OF THE DUCTLESS GLANDS.

CHAPTER CXVII.

DISEASES OF THE SUPRARENAL BODIES.

I. HISTOLOGY AND FUNCTIONS OF THE SUPRARENAL BODIES.

General Description.—The suprarenal bodies are 'endocrine organs', ductless glands with an internal secretion. Are in anatomical but no other relation to kidneys; do not move with a *rena mobilis*. Average weight 5 to 7 grm. Consist of a fibrous capsule, and within this two layers, (1) cortex, (2) medulla, entirely distinct in origin and function, though with same blood-supply.

CORTEX.—Yellow colour, with browner band next to medulla. Epithelial origin. Connective tissue passes in from capsule. *Histology*: Roundish cells arranged in strands. Three layers recognizable: (a) Zona glomerulosa. (b) Zona fasciculata: definite columns; many lipoid granules. (c) Zona reticularis: junction of columns; cells pigmented.

MEDULLA.—Soft and dark-red (from blood present). Consists of: (a) Anastomosing strands of cells enclosing blood-spaces; origin similar to sympathetic ganglia, and hence ectodermal; stain brown with chromic acid, hence called '*chromaffin cells*'. (b) Nerve cells like sympathetic ganglion cells; single or in small groups. (c) Non-medullated nerve-fibres. Blood-vessels very numerous. Nerve-supply rich, chiefly from solar and renal sympathetic plexuses, and some fibres from [illegible] us.

'Chromaffin Tissue or Cells' in other Sites.—Kohn's 'paraganglia', or 'chromaffin bodies' are small masses, not exceeding a pea in size, along the aorta, chiefly the abdominal aorta and near kidneys—e.g., 'Zuckerkandl's organ' near origin of superior mesenteric artery. Structure roughly resembles suprarenal medulla: many cells chromaffin: probably same function. Kohn also included: (1) Carotid glands, at bifurcation of common carotid: contain chromaffin cells: possibly also relationship to parathyroid. (2) Coccygeal glands: probably sympathetic origin, but chromaffin cells doubtful.

Accessory Suprarenal Bodies. Suprarenal 'Rests'.—Consist of cortical substance. Common in the liver and other structures—e.g., 'Marchand's organ' in broad ligament near ovary (almost constant). Rarely with both cortex and medulla, occasionally in solar plexus.

Functions of the Suprarenal Bodies and Chromaffin Tissue.—

MEDULLA AND CHROMAFFIN TISSUE.—The active principle

Suprarenal Bodies —Functions, *continued.*

has been isolated, known as 'epinephrin' or 'adrenalin': the latter is prepared synthetically. All chromaffin tissue has same function.

MAIN ACTION OF ADRENALIN.—(1) *Constricts peripheral blood-vessels.* Coronary arteries dilate. Pulse slows and then increases. (2) *Raises blood-pressure.* (3) Produces hyperglycæmia and glycosuria: transient. In animals, experimentally, produces an arteriosclerosis.

The medulla, and chromaffin tissue, also connected with pigmentation of the skin, and possibly with functions of muscles.

Amount is diminished in certain acute diseases, e.g., diphtheria; also in Addison's disease.

CORTEX.—Function little known. Influences *sexual activity, growth,* and pregnancy. (*See* TUMOURS, p. 728.)

Relations of Suprarenal Bodies to other Glands, etc.—Doubtful.

THYROID.—Pigmentation may occur in Graves' disease.

PANCREAS.—Possibly control carbohydrate metabolism of liver in opposite directions.

KIDNEY.—Hyperplasia of chromaffin tissue frequent in renal disease.

PREGNANCY.—Hyperplasia of cortex occurs.

II. ADDISON'S DISEASE.

A rare condition characterized by progressive weakness, muscular and cardiovascular, by gastro-intestinal disturbances, and by pigmentation of the skin, resulting from disease of the suprarenal bodies and of the chromaffin tissue.

Etiology.—

AGE.—Wide limits: commonest between 20 and 40 years.

SEX.—Males in some excess.

No relation to heredity, race, or other disease except tuberculosis

Morbid Anatomy.—

LESIONS OF THE SUPRARENAL BODIES—

1. TUBERCULOSIS.—In great majority: other causes very rare. Usually advanced caseation, bilateral, and secondary to tuberculosis elsewhere, e.g. lungs.
2. ATROPHY.—Simple, or with chronic fibrosis
3. MALIGNANT DISEASE, hæmorrhages, hydatid disease, etc.

CHROMAFFIN TISSUE IN PARAGANGLIA is usually involved with the suprarenals. Exceptions rare: may account for disease of suprarenals without Addison's symptoms. Also very rarely the converse—viz., suprarenals normal, paraganglia affected by pressure or inflammation, and symptoms present.

OTHER ORGANS.—Changes slight. Thymus often persistent. Heart in brown atrophy. May be tuberculosis elsewhere.

Symptoms.—

ONSET.—Insidious; rarely acute. Initial symptom is usually *weakness*, muscular and general. Often months before symptoms become characteristic.

CHARACTERISTIC SYMPTOMS.—

1. ASTHENIA.—Extreme and progressive, muscular and cardiovascular (*see* 4), disproportionate to wasting.
2. GASTRO-INTESTINAL DISTURBANCES.—Variable; may be absent until late stage; remissions common. *Anorexia* marked. Nausea. Attacks of obstinate *vomiting*. Constipation early; later may be diarrhœa.
3. PIGMENTATION OF SKIN.—*Colour*: Light brown to deep brown or almost black. *Distribution*: On parts: (*a*) Exposed; (*b*) Normally pigmented; (*c*) Exposed to irritation, e.g., waistband; (*d*) Mucous membranes—here usually patchy. No itching. Occasionally: deeply pigmented spots; leukodermia. Pigmentation rarely absent.
4. LOW BLOOD-PRESSURE AND CARDIAC WEAKNESS.—*Systolic blood-pressure* 70 to 90 mm. Hg. Pulse feeble. Giddiness and syncope common.

OTHER SYMPTOMS.—*Wasting*, but not extreme emaciation. *Anæmia* rarely marked. *Temperature* often subnormal. Headache and pain in the back: occasionally neuralgias. 'White line' after scratching skin: often definite, but value slight. *Urine*: no changes: glucose tolerance not reduced. Glucose in blood diminished.

Progress and Termination.—Asthenia progresses. Death occurs from weakness, sudden cardiac failure, and occasionally general tuberculosis.

DURATION.—Usually one to three years. Rarely, a few months; or up to ten or more years.

RECOVERY recorded in a few apparently authenticated cases.

Diagnosis.—Based on characteristic symptoms, *asthenic group* being essential. Note absence of cause, of any marked anæmia, and of other factors producing pigmentation. Mucous membranes escape pigmentation in other conditions, except rarely in forms due to intra-abdominal disease. *Neurasthenia* may simulate the disease closely, especially with pigmentation of a multipara. Dark-skinned races difficult.

℞ LEYTON'S TEST.—Administer adrenal extract gr. iij. t.d.s. for three days per os; a rise of 10 mm. in blood-pressure suggests adrenal insufficiency

Treatment.—

1. PALLIATIVE.—Rest and warmth. Symptomatic treatment for cardiac and gastric disturbances.
2. SUPRARENAL THERAPY.—In experimental stage, but should be tried. Various methods, e.g.: (*a*) Fresh sheep's glands, one or more daily; (*b*) Adrenalin solution (1–1000) ♏v, t.d.s., increasing to ♏xx to xxx.

PIGMENTATION OF THE SKIN.

Common causes of diffuse pigmentation are few. In a number of conditions it occurs rarely, or a sallow skin causes confusion, or pigmentation is patchy

Commoner Causes.—

1. CHRONIC IRRITATION OF SKIN - 'Vagabond's discoloration' From lice and dirt Many scratches
2. INTRA-ABDOMINAL CONDITIONS (a) Tuberculosis, Addison's disease. (b) Cancer, especially of *peritoneum* (frequently) (c) Dilatation of stomach Rarely in gastric ulcer.
3. ARSENIC
4. PREGNANCY —Especially affects face. transient. Occasionally in uterine disease
5. SKIN CONDITIONS, e g, leucodermia

Rarer Causes. —Exophthalmic goitre Malaria Pernicious anæmia usually, if not invariably, from therapeutic arsenic. Lymphadenoma

Various, Rare, or More Localized Conditions.—Hæmochromatosis. Melanotic neoplasms Argyria Pellagra localized Ochronosis Von Recklinghausen's disease

Conditions which may give rise to Difficulties in Diagnosis.—Neurasthenia Chronic constipation Chronic jaundice, or acholuric family jaundice Chronic cardiac and renal conditions

III. TUMOURS OF THE SUPRARENAL GLANDS.

Classification is confused by frequent difficulty in deciding whether (a) Origin is from suprarenal or from liver, or sometimes from kidney, (b) Tumour is carcinoma, sarcoma, or other form whether benign or malignant

1. BENIGN TUMOURS Adenoma or benign hypernephroma Small tumours common, occasionally large and hæmorrhagic
2. MALIGNANT TUMOURS —*Carcinoma or sarcoma* differentiation often very difficult. may resemble adenoma of cortex Origin either from cortex or medulla Yellowish colour, but hæmorrhage and necrosis frequent Tend to invade vena cava Occur at any age

   METASTASES —Very frequent Common sites (a) *Liver* (b) *Lungs* (c) *Bones*: skull, vertebræ, etc (cf THYROID TUMOURS). (d) Kidneys Lymphatic glands
3. TUMOURS OF ACCESSORY SUPRARENAL GLANDS OR 'RESTS' (*Grawitz's tumour*).—Malignant hypernephroma of kidney. Now considered to be a true renal adenoma or adenolipoma. (*See* TUMOURS OF THE KIDNEY, p. 594)

Clinical Characteristics of Suprarenal Tumours.—

SIGNS AND DIAGNOSIS —*Mobile Rapid growth.* Often indistinguishable from renal tumours. No hæmaturia. Metastases

may suggest diagnosis. Fatal in few months after becoming obvious.

SYMPTOMS.—

1. IN ADULTS.—Addison's disease not definitely produced. No pigmentation.
2. IN CHILDREN.—
   a. Remarkable group with *precocity, general and sexual*, excessive hair, increased fat, may be pigmentation. Females predominate in number, but symptoms most marked in males, in whom extreme mental precocity and great muscular strength may occur (Bulloch and Sequeira). A sub-group occurs in both sexes, with greater obesity, but other changes slight, *except growth of hair*. Group suggests an internal secretion of cortex influencing general and sexual development. *Progeria* may be the converse
   b. Group with suprarenal and cranial tumours and exophthalmos (R. Hutchison). From metastases.

Hypoplasia and Hyperplasia of Suprarenal Glands.—Absence occurs in monsters, especially with skull defects Hypoplasia is described in osteogenesis imperfecta and osteomalacia Hyperplasia of cortex occurs in chronic nephritis.

CHAPTER CXVIII.

DISEASES OF THE THYMUS GLAND.

I. FUNCTIONS OF THE THYMUS GLAND.

Functions unknown. Following extirpation, results are variable and doubtful, may be none, claims include conditions resembling rickets, cachexia, hypertrophy of testis. Evidence of internal secretion slight. Possibly a lymphoid organ

II. HYPERTROPHY AND 'PERSISTENCE' OF THYMUS.

At birth thymus weighs about 7 grm. Maximum, at two years, about 10 grm Atrophy then commences, and after puberty is more rapid. Thymus over 15 grm is thus not only 'persistent', but *enlarged* (Warthin). Occurs up to 50 and rarely 70 grm.

Occurrence.—Enlargement of the thymus occurs in: (1) Status lymphaticus (2) Occasionally in Graves' disease, Addison's disease, myasthenia gravis, and other diseases. (3) In conditions affecting lymphoid tissue: acute infections, leukæmia, etc.

Situation of Enlargement.—Mainly behind sternum; may extend over pericardium. Rarely, recognized in life by dull area and by X rays,

Hypertrophy and 'Persistence' of the Thymus, *continued.*

Morbid Anatomy.—Tissue soft. *Histology*: Little change from normal; chiefly hyperplasia of medullary portion; Hassall's corpuscles somewhat large and numerous.

Symptoms.—Three groups (Warthin), confined to children.

1. THYMIC STRIDOR.—Congenital or arising soon after birth. Apparently indistinguishable from 'congenital laryngeal stridor'
2. THYMIC ASTHMA (*Kopp's asthma*).—Severe asthmatic attacks May occur in acute infections, especially pulmonary, bronchitis, etc. Tracheotomy useless unless long tube passes thymus. Frequently fatal. Analogous to 'laryngeal spasm'
3. THYMIC DEATH.—*See* STATUS LYMPHATICUS.

III. STATUS THYMICO-LYMPHATICUS.

(*Status Lymphaticus.*)

A condition, occurring in flabby children, characterized by enlargement of the thymus and lymphoid tissue, and usually recognized at autopsy following sudden death from trivial causes.

Pathology.—

Hyperplasia of: (1) Thymus. (2) Lymphatic glands; especially intra-abdominal; no *large* masses, *external glands only slight.* (3) Tonsils Adenoids usually present

Spleen occasionally enlarged, also solitary follicles and Peyer's patches.

Signs of asphyxia often present. Dilatation of left ventricle common.

Symptoms.—Usually a fat, somewhat pale, but apparently healthy child. Generally no previous suggestive symptoms, infrequently, some asthmatic attacks. *Sudden death* follows a trivial cause e.g., at commencement of anæsthesia for tonsillectomy.

Theories of Mechanism of Thymic Death, and of Thymic Asthma and Stridor.—

(1) MECHANICAL, FROM PRESSURE OF ENLARGED THYMUS.—Compression of trachea has been found at autopsy, but is frequently absent; yet possibly compression may have occurred during life. Cardiac failure from compression of vessels or nerves by acute hyperæmic gland is sometimes assumed Cure of asthma and stridor is reported after thymectomy.

(2) TOXÆMIC ACTION FROM INTERNAL SECRETION.

Neither theory is proved, but space between sternum and vertebræ is very small, and *pressure* theory is reasonable.

Diagnosis.—Status lymphaticus occasionally suggested in children of type and pathological condition described. Examine for thymic dullness, and radiograph. If suggestive, care in anæsthesia, etc, necessarily extreme.

Treatment.—General tonics. Condition does not extend beyond puberty. Thymectomy inadvisable.

IN ACUTE CONDITION.—Artificial respiration; tracheotomy if long tube available; possibly thymectomy or thymotomy.

CHAPTER CXIX.

DISEASES OF THE THYROID AND PARATHYROID GLANDS.

DISEASES OF THE THYROID GLAND.

I. CONGESTION.

At sexual periods—i.e., puberty, menstruation, and possibly coition—the gland may enlarge, causing temporary discomfort.

II. THYROIDITIS.

Inflammation, simple or acute suppurative. Both very rare, especially latter. Secondary to sepsis elsewhere or acute infectious disease. *Symptoms*: Acute local and constitutional signs of sepsis.

III. TUMOURS OF THE THYROID.

Benign.—*Adenoma* Encapsuled tumour: usually in right lobe.

TYPES.—(*a*) Resembles fœtal gland: alveoli filled with spheroidal cells, no colloid; small and single. (*b*) Colloid adenoma: resembles developed gland, but epithelium flattened, alveoli large, and much colloid; often multiple; cysts and hæmorrhage common; *frequently occurs with parenchymatous goitre.*

Malignant.—Usually in pre-existing goitre; commonly at menopause.

TYPES.—(*a*) Adenocarcinoma: often very slow. (*b*) Carcinoma. (*c*) Sarcoma: rare; rapid.

HISTOLOGY. All stages from typical carcinoma to rare forms resembling normal gland. Macroscopically all varied—e.g., like 'colloid goitre'.

SYMPTOMS.—Growth surrounds vessels and structures, while goitres displace them: hence *early fixation*, and marked pressure symptoms—dysphagia, dyspnœa, cough, hoarseness, congestion and œdema of face, inequality of pupils, etc. Perforation into trachea or œsophagus not common. In later stages, may be adhesion to skin, enlargement of lymphatic glands. Myxœdema has occurred.

METASTASES.—*Frequent.* Primary growth often small, discovered after metastases or at autopsy.

SITES.—(*a*) Bones: usually skull, jaw, long bones, sternum, pelvis. (*b*) Lungs. Occasionally liver and kidneys.

Histologically, all degrees, from almost normal thyroid tissue to carcinoma not suggesting thyroid. May pulsate.

Tumours of Aberrant Thyroid.—Lingual thyroid: *Central swelling at back of tongue*; not uncommon; from remains of thyroglossal duct; cysts and hæmorrhages common; interferes

Tumours of the Thyroid, *continued.*

with swallowing; removable by operation; thyroid gland may be absent, and myxœdema follow. Rarely in other sites. Tetany never follows removal of a lingual thyroid, owing to position of parathyroids.

Tuberculosis, Syphilis, etc.—Very rare.

Parenchymatous, Colloid, Fibrous Goitres, etc.—*See* GOITRE.

IV. GOITRE.

(*Bronchocele.*)

A chronic general enlargement of the thyroid gland, occurring endemically or sporadically, of unknown origin, and producing symptoms by pressure.

Distribution.—Sporadic cases occur in all parts. Endemic foci in mountainous or hilly districts: Switzerland—very prevalent; France, Germany, and Austria in mountainous regions—e.g., Tyrol, Styria, Pyrenees; England—Derbyshire ('Derbyshire neck'); Central Asia; Himalayas; Japan.

Etiology.—

AGE.—Commences most commonly at puberty, less after 20 years, rarely after 40 years. A goitre, once present, may continue to grow steadily, or suddenly increase.

SEX—Females 6 to males 1. Ascribed to relation of thyroid to sexual organs—e.g., congestion during menstruation, pregnancy, and coition.

CONGENITAL.—Rare, except in endemic areas and with goitrous parents.

Cause.—Due to absence from or probably presence in *water* of some unknown substance: *boiled* water is harmless. Endemic areas and 'goitre springs' and 'wells' exist: persons and animals become goitrous, and recover on leaving district or boiling water. Theories as to the responsible factor in the water include: (1) Poverty in iodine; (2) Hardness; (3) Radio-activity—high in certain waters. Endemic areas roughly but not invariably correspond to certain geological formations. (4) McCarrison, in Kashmir, found that the specific agent was removed by boiling, stopped by Berkefeld filter, and that the residue on the filter produced goitre, but was harmless if boiled. He attributed the cause to contamination of the water by pathogenic organisms from the intestinal tract (not confirmed).

Morbid Anatomy.—Entire gland usually enlarged: occasionally one lobe (right) or isthmus. May become enormous.

'*Parenchymatous Goitre*' is the term applied to general enlargements. *Small* goitres appear almost normal, but histologically there is always: (1) Increase in colloid; (2) Proliferation of epithelium. In *large* goitres: epithelium flattened, alveolar walls thin, great increase in colloid, alveoli vary in size. *Blood-vessels* in capsule increased, but scanty in the tissue.

DEGENERATIONS are very common Various types Often several forms in same gland (1) *Colloid degeneration* ('colloid goitre') (2) *Hæmorrhage*: Size of gland increases rapidly (3) *Cysts*: Contents watery, viscoid, or hæmorrhagic, papillary ingrowths common (4) *'Fibrous Goitres'* Much fibrous tissue, common in old goitres from inflammation Hyaline degeneration, necrosis, rarely calcification, may occur

Encapsuled adenomata common

Symptoms.—Attention first attracted by size of neck Any thyroid causing a swelling on neck is enlarged Symptoms result from *pressure* Are produced by (1) Very large glands (2) *'Sub sternal goitres'*, passing behind sternum dangerous even when small (3) Occasionally small goitres surrounding trachea *Effect on trachea* chiefly compression laterally Other structures are more often displaced than compressed

The characteristic symptoms are (1) *Dyspnœa* The predominant symptom, occurs particularly at night May be paroxysmal and fatal (Recurrent laryngeal nerve) (2) Inspiratory stridor and cough, often no sputum (Tracheal pressure)

Other symptoms are not common, and if marked suggest neoplasm Occasionally Dysphagia, congestion of veins of neck (œdema rare), hoarseness Cardiac affections 'goitre heart', are not uncommon, usually described as mechanical due to pressure on vagus or to dyspnœa, but often suggest some degree of hyperthyroidism

Sudden increase in size suggests hæmorrhage or neoplasm.

Prognosis.—Small, soft, and early goitres, and those in young people, often recover on medical treatment or on leaving goitrous district, but not old or fibrous forms Operation for pressure symptoms

Diagnosis. The thyroid gland moves upwards on swallowing.

In parenchymatous goitre the gland is usually freely movable displaces vessels and structures rather than encircles them as do neoplasms No symptoms of exophthalmic goitre, thrills and murmurs over swelling rare With sternomastoid rendered tense, gland is felt passing deep to it Lower border *usually* above clavicle

Treatment.—

PROPHYLAXIS Boil water in goitrous districts

MEDICAL TREATMENT —

IODINE —Give pot iodide gr v, t d s, by mouth, for two or three weeks, then one week interval, continue two to three months Results very good Iodine application to gland (less effective) Ung. hydrarg. iodidi rubri in half strength, alternate nights Iodine injections into gland inadvisable, many deaths (from injection in blood vessels)

Note —Enlargement of gland suggests a hypertrophy resulting from increased demands from the body. Iodine stimulates activity of gland. During treatment, hyperthyroidism may occur—viz., rapid pulse, etc.—

Goitre—Treatment, *continued.*

an 'iodism' differing from the ordinary iodine irritation of mucous membranes.

THYROID EXTRACT.—Occasionally effective. Inferior to iodine.

X RAYS TO GLAND.—Good results recorded.

SURGICAL TREATMENT.—Indicated for dyspnœa, substernal goitres, and swellings not reacting to iodine. Portion of gland removed.

V. HYPOTHYROIDISM.

(*Myxœdema and Cretinism.*)

Clinical Types.—(1) *Myxœdema*; (2) *Cachexia strumipriva*—following thyroidectomy; (3) *Cretinism*, sporadic and endemic.

1. Myxœdema.

A condition due to deficiency of the thyroid secretion, characterized clinically by defective metabolism and mental changes, and pathologically by atrophy and fibrosis of the thyroid gland.

Etiology.—

AGE.—Onset most frequent from 30 to 50 years.

SEX.—Females 6 to males 1. No special relation to sexual functions.

HEREDITY.—No obvious influence.

EXCITING CAUSE of the fibrosis and atrophy of gland.—Unknown. Neither alcohol nor syphilis. Rarely exophthalmic goitre precedes, fibrosis occurring in the hypertrophied gland.

Morbid Anatomy.—

THYROID GLAND.—Fibrosis and atrophy: weight often 3 to 5 grm. instead of 30. Enlarged rarely.

SUBCUTANEOUS ŒDEMA.—Presence of excessive mucin formerly described, but is doubtful. Explanation of the swelling unsettled: possibly a form of granulation tissue.

OTHER CHANGES (not constant).—Enlargement of hypophysis. Myocarditis and arteriosclerosis (in advanced types).

Symptoms.—Characterized by *slowness* in all functions, mental, muscular, and metabolic.

ONSET insidious. *Early complaints*: Increased bulk, languor, coldness.

'SOLID ŒDEMA'.—Characteristic swelling of subcutaneous tissues; does not pit on pressure. Increases bulk and alters appearance. Distribution general, but marked where tissues lax, e.g., 'supraclavicular pads'.

PHYSIOGNOMY.—Expressionless features, broad and bloated. Eyelids puffy and drooping. Lips and nostrils thick. General yellow tint, with red patch on cheek ('strawberries and cream'). Appearance usually characteristic.

SKIN.—Dry and rough. No sweating. *Hair* sparse and dry.

GAIT AND ALL MOVEMENTS.—Slow and deliberate. *Hands and feet* large and flat.

MENTAL CONDITION.—Cerebration slow. Memory defective. Speech slow and muffled. Deafness not uncommon. Often irritable. Headache. Rarely visual and other hallucinations and finally dementia.
ALWAYS COLD better in warm weather. CONSTIPATION. ANÆMIA moderate.
PULSE slow and regular. When disease advanced, sometimes chronic myocarditis.
TEMPERATURE low.
URINE.—Slight albuminuria not uncommon, rarely glycosuria.
THYROID GLAND impalpable.
MENSTRUATION irregular. STERILITY not invariable.
BASAL METABOLISM reduced 25 to 40 per cent.

Clinical Varieties.—Incomplete and mild forms occur. Exophthalmic goitre may pass into myxœdema and symptoms be temporarily combined as gland atrophies, or very rarely a mixed condition occurs from onset.

Progress in Absence of Treatment.—Slowly progressive over many years. Death from intercurrent disease, tuberculosis, myocarditis, or nephritis.

Diagnosis.—Simple in marked cases. Early diagnosis now expected. Diagnosis from:
CHRONIC NEPHRITIS. Resembles myxœdema in pallor, swelling and albuminuria. Differs in absence of solid œdema, dry skin and hair, changed mental condition.
OBESITY. Sweating marked and patient prefers cold weather. Differs from 'solid œdema'.

Treatment.—
PREPARATIONS OF THYROID GLAND (*see* CRETINISM).—Initial dose, gr. xv daily. Final dose usually about gr. v daily.
PROGRESS UNDER TREATMENT. (1) *Loss of weight* best measure of dosage; often loss of 2 to 4 stone. (2) Symptoms disappear and recovery almost complete—permanent if thyroid continued.
THYROIDISM.—If dosage excessive tachycardia and nervousness may develop; reduce dose.
MYOCARDITIS.—Keep in bed at onset of treatment if cardiac symptoms present.
RELAPSES on omitting thyroid are equally controlled on resumption.
GENERAL TREATMENT.—*Warmth*. Full and nutritious diet, etc., important.

2. Cachexia Strumipriva or Thyreopriva.

Following operative removal of thyroid. Symptoms of myxœdema occur in 15 to 30 per cent., may commence in one to two weeks. Amenable to thyroid treatment. Occasionally tetany and coma, if parathyroids injured.

Hypothyroidism, *continued.*

3. Cretinism.

A chronic but curable condition commencing in infancy, due to deficiency of the thyroid secretion, characterized clinically by defective development mentally and bodily, and pathologically by absence or disease of the thyroid gland.

Varieties.—Two forms occur, essentially the same (1) Sporadic, (2) Endemic

1. SPORADIC CRETINISM --

ETIOLOGY —Females 60 per cent No known factors

MORBID ANATOMY —

Thyroid Gland —(1) Absent most common (2) Advanced fibrosis and atrophy less common (3) Goitre very rare (cf ENDEMIC CRETINISM)

Skeletal changes Growth arrested, bones thick and short Rough resemblance to rickety bones, but epiphyseal cartilages show, not proliferation, but deficient growth and delayed ossification

Thymus often persistent Hypophysis sometimes enlarged No visceral changes

SYMPTOMS —Rarely noticed before six months, then deficient growth, mental dullness, large tongue, and dry skin By second year symptoms definite, subsequently develop fully

Growth stunted—Full grown cretin rarely exceeds 4 feet

Bodily proportions abnormal—Head large, neck short breast flat vertebræ curved Hands and feet 'spade-like' Abdomen prominent, umbilical hernia common Fontanelles close late

Œdematous appearance does not pit *Anæmia* moderate

Physiognomy —*Face* broad and puffy *Eyes* wide apart, lids swollen *Nose* flat *Alæ nasi* thick nostrils wide open *Tongue* protruding, mouth open, dribbling common

Skin dry sweating rare *Hair* coarse and scanty. *Teeth* dentition delayed, early caries *Nails* brittle

Sexual organs small

Mental condition apathetic—Stolid, but easily amused, infrequently, vicious Speech, very slight Deafness common In advanced stages, may be imbecile.

Muscular weakness marked

Always cold *Constipation* *Temperature* often subnormal

VARIATIONS – 'Juvenile myxœdema'. onset in childhood. Cretinism with goitre In both forms, symptoms usually partial

2. ENDEMIC CRETINISM —Occurs where goitre is prevalent; etiology similar. When a family enters goitrous district, goitre appears in first generation, and cretinism in later ones.

MORBID ANATOMY —*Goitre* present at birth in 60 per cent. Histology: atrophy and fibrosis.

SYMPTOMS.—As in sporadic type.

Diagnosis.—Simple in marked cases From —

MONGOLIAN IDIOCY —Eyes placed obliquely Restless No subcutaneous thickening Often later child in large family No improvement with thyroid extract

Other difficulties may be Mental deficiency Infantilism Rickets

Treatment. –

PREPARATIONS OF THYROID GLAND —Many forms efficacious e g, dried gland in tablets, thyroideum siccum, or liquor thyroidei Two stages in treatment (1) To cure the disease (2) To prevent recurrence Treatment must continue for life For each subject correct dosage found by experiment and progress as below Commence with thyroideum siccum gr j daily (or equivalent in fluid) and increase until efficient, rarely exceeds gr iij With continuation of excessive dosage, hyperthyroidism occurs viz nervousness tachycardia etc

PROGRESS UNDER THYROID TREATMENT (1) *Initial loss of weight*, from reduction of œdema then gain as growth proceeds (2) *Growth in height* often several inches in first six months (3) *General mental and bodily development* tending to removal of all symptoms of condition may be complete if diagnosis early degree of recovery varies inversely with age at commencing treatment if over ten years child tends to remain younger than his age and mental condition may be impaired

GENERAL TREATMENT Fresh air good diet, care of skin etc, important in early stages

If untreated, life rarely exceeds 30 to 40 years

VI. EXOPHTHALMIC GOITRE.

(*Graves, Basedow's or Parry's Disease*)

A disease characterized clinically by enlargement of the thyroid gland exophthalmos tachycardia and tremor and pathologically by overactivity of the thyroid gland

Etiology. –

SEX At least 10 females to 1 male

AGE Usually between puberty and the menopause

HEREDITARY INFLUENCES —Often several members of one family affected Hysteria and nervous conditions common in family

EXCITING CAUSES —Unknown Sudden onset may follow a shock or acute illness (note resemblance of symptoms to those of terror)

Morbid Anatomy.—

THYROID GLAND —General enlargement Superficial vessels are large and distended Substance soft Cut surface lacks gelatinous appearance

HISTOLOGY —*Great increase in parenchymatous cells and supporting tissue, and absence of colloid* Alveoli of various sizes projections of lining membrane and epithelium run into lumen, many cells desquamated. Changes usually diffuse, less often focal. Gland in condition of great secretory

Exophthalmic Goitre—Morbid Anatomy, *continued.*

activity. In severe cases appearance not unlike sarcoma. *Rarely*, in severe cases, colloid increased.

In later stages, fibrosis increased, and there may be areas of atrophy.

Iodine Content in Thyroid Gland.—Much reduced, often almost absent; but greatly increased in cases in which colloid is present. Note that: (*a*) Iodine always depends on presence of colloid; (*b*) Absence of iodine is explicable by rapid absorption of colloid into circulation.

THYMUS.—Persistent, extending to or over pericardium. Structure normal.

EXOPHTHALMOS.—Only change is increased fat in orbit.

Cervical glands always enlarged at operation or autopsy.

No constant changes in sympathetic ganglia, parathyroids, hypophysis, or central nervous system.

Pathogenesis.—*Excessive formation and absorption of secretion of the thyroid gland—i.e., hyperthyroidism.* Supported by:—

1. Symptoms are antithesis of myxœdema and cachexia strumipriva (hypothyroidism).
2. Thyroid administration in excess (e.g., for obesity) produces similar symptoms—except exophthalmos in man (observed, however, in monkeys and dogs).
3. Thyroid administration aggravates exophthalmic goitre.
4. Symptoms improve on removal or reduction of gland.
5. Gland is in condition of secretory activity: resembles small portion left in animals after experimental removal.

Symptoms.—

ONSET.—Usually gradual. Main symptoms may arise simultaneously or in sequence. Rarely, onset acute: cardiac symptoms severe, often fatal.

CHARACTERISTIC SYMPTOMS.—(1) Enlargement of the thyroid; (2) Tachycardia and circulatory disturbances; (3) Exophthalmos; (4) Tremor. Other important symptoms are nervousness, sweating, wasting, headache, anæmia. Patient complains of one or more of these (rarely of tremor).

1. ENLARGEMENT OF THE THYROID.—Uniform, or right lobe larger than left. Rarely very large. Soft. No tenderness.

   Inspection.—Often visible pulsation, from vascularity, or transmission from carotids.

   Palpation.—Thrill not uncommon.

   Auscultation.—Loud murmur, systolic, double, or continuous (*bruit de diable*).

   Pressure symptoms very rare. Size may vary from time to time: not always in relation to severity of symptoms.

2. TACHYCARDIA AND CIRCULATORY DISTURBANCES.—

   Tachycardia.—Pulse 100–160, regular. Most constant symptom. Rapidity easily increased. *Palpitations* common.

Cardiac signs.—Area of pulsation increased and forcible. Heart-sounds very loud. Murmurs rarely absent: apical systolic or at base, especially 2nd left space.

Visible pulsation in arteries in neck.—Often extreme. Also pulsation in peripheral vessels and veins, and flushing of head and neck.

In severer forms, cardiac dilatation (may be acute) or hypertrophy occurs.

Blood-pressure variable, often high.

3. EXOPHTHALMOS AND OCULAR SYMPTOMS.—

Exophthalmos.—Staring expression. May be unilateral. Eyeballs protruded, lids retracted Cause unknown, possibly spasm of orbital muscle of Müller or increased intraorbital fat. Usually last symptom to disappear. *Vision normal.*

Von Graefe's sign.—On looking down, upper eyelid lags or descends in jerks, sclerotic becoming visible.

Stellwag's sign.—Wide palpebral fissure; spasm or levator palpebræ superioris.

Mœbius' sign.—Lack of convergence for near objects. No diplopia.

Joffroy's sign.—On looking upwards, no wrinkling of occipitofrontalis.

4. TREMOR.—Fine. Involuntary. Affects whole extremity. Often unnoticed by patient.

OTHER SYMPTOMS, rarely absent.—*Nervousness*; irritability; *very rarely*, acute mania (usually fatal). *Sweating*, and flushing; dislikes heat, better in cold. *Wasting*: serious symptom when severe. *Headache*: often connected with vascular throbbing. *Anæmia* and irregular menstruation. Reflexes brisk.

VARIOUS AND OCCASIONAL SYMPTOMS.—Slight *pyrexia*. Infrequent winking. *Pigmentation* of skin. Vomiting, less often diarrhœa. Urine: albuminuria and glycosuria not uncommon. Blood: relative lymphocytosis (often absent).

Progress.—Condition tends to advance to a maximum in about a year. About 25 per cent die directly from the disease: either gradually from weakness or cardiac failure, or rarely from sudden syncope. About 50 per cent practically recover. Remainder become chronic. 'Relapses' common: usually incomplete recovery in interval. Myxœdema may gradually develop. *Prognosis* bad with: severe wasting, irregular pulse, persistent vomiting or diarrhœa, delirium.

Diagnosis.—Usually simple on inspection—viz., exophthalmos and prominent thyroid. Incomplete forms difficult. Never diagnose with normal pulse-rate. Hysterical tremor with rapid pulse difficult to distinguish.

Treatment.—

1. MEDICAL TREATMENT.—Rest absolute. Diet moderate. Leiter's tubes or ice-pack to neck.

DRUGS.—Tinct. belladonnæ. Liquor arsenicalis. Commence.

Exophthalmic Goitre—Treatment, *continued*

with small dose, increase until signs of toxicity, intermit, use for long periods Pot. bromide if patient very nervous

2. X RAYS to thyroid gland —Promising results Combine with rest and medical treatment.

3. SURGICAL TREATMENT —Operation should remove portion of gland tissue *Results good* in cases of medium severity If relapse, second operation is practicable

OPERATIVE RISKS FROM (a) Hyperthyroidism subsequently attributed to great temporary absorption of secretion Extreme tachycardia and delirium (b) Anæsthetic badly borne (c) Tetany from removal or interference with parathyroids In selected cases, immediate mortality not above 5 per cent

CONTRA INDICATIONS —Pulse over 130 irregularity of heart marked prostration Possibly marked relative lymphocytosis (Kocher) persistent albuminuria or glycosuria

SPECIFIC TREATMENT —Various preparations —e g (a) 'Rodagen'—milk of thyroidectomized goats (b) Cytolytic serum from animal injected with thyroid gland. Results at present unconvincing

DISEASES OF THE PARATHYROID GLANDS.

The parathyroid glands, commonly four in number, are arranged in two pairs, external and internal, along the posterior inner border of the lateral lobes of the thyroid Size, not exceeding ½ inch in length and ¼ inch in breadth Brown in colour In man, never included in thyroid gland Origin from epithelium in 3rd and 4th branchial clefts Are *ductless glands*, with an internal secretion

Total removal results in fatal convulsions

Symptoms —Following removal of at least two glands or damage to circulation *tetany* Onset in two to five days (For symptoms, *see* TETANY) In cases subsequently surviving, principal symptoms are (1) Wasting, (2) Diminished carbohydrate tolerance (3) Increased excretion of calcium

Relation to other Spasmodic Conditions, e g, tetany, laryngismus infantile convulsions Doubtful A syndrome, 'spasmophilia', has been described

CHAPTER CXX

DISEASES OF THE SPLEEN.

I. FUNCTIONS OF THE SPLEEN.

In the Fœtus the spleen forms red blood cells

During Life the functions are :—

Haemolysis, i.e., destruction of red cells

Formation of lymphocytes, a function common to all lymphoid tissue.

3. An unknown response to infections, probably defensive; the spleen usually enlarges, and contains many bacteria.
4. In anæmia, a possible reversion to formation of red cells.

Splenectomy.—Removal of normal spleen is followed by—

1. No serious sequelæ: hence the organ is not necessary to normal life.
2. No metabolic disturbance, nor symptoms suggesting an internal secretion
3. Blood changes: (a) Polynuclear leucocytosis (20,000–40,000) fever usual, often suggesting sepsis (b) Temporary anæmia. (c) After many months, a relative lymphocytosis. (d) Enlargement of hæmolymph (prevertebral) glands

Similar changes follow removal of many abnormal spleens

Removal of Abnormal Spleens.—Operative risk depends mainly on size of spleen Before operation, size should if possible be reduced by X ray and other treatment. Contra indicated in pernicious anæmia and leukæmia, course being unaffected by removal. Indicated in splenic anæmia group and acholuric jaundice

II. ENLARGEMENT OF SPLEEN: SPLENOMEGALY.

Enlargement of the Spleen occurs with.—

1. DISEASES OF THE BLOOD*—(a) Leukæmia (b) Splenic anæmia and Banti's disease, (c) Pernicious and aplastic anæmia, (d) Erythræmia, (e) Acholuric family jaundice, (f) Von Jaksch's anæmia
2. INFLAMMATIONS AND SPECIFIC INFECTIONS—(a) Sepsis—e g, septicæmia, infective endocarditis, (b) Specific fevers, especially enteric Also (c) Infarcts
3. CONDITIONS ASSOCIATED WITH HEPATIC CIRRHOSIS—(a) Alcoholic cirrhosis, (b) Hanot's hypertrophic cirrhosis, (c) Hæmochromatosis (Also Banti's disease)
4. HODGKIN'S DISEASE
5. TUBERCULOSIS
6. SYPHILIS } Great enlargement occurs in a small proportion.
7. MALARIA }
8. RICKETS
9. KALA AZAR

Rare—

10. SPLENIC AND PORTAL VEIN OBSTRUCTION.
11. GAUCHER'S SPLENOMEGALY.
12. AMYLOID DISEASE

Great Enlargement. Common causes are: (1) Chronic leukæmia, (2) Splenic anæmia, (3) Syphilis, (4) Malaria; (5) Kala-azar. Rare diseases with great enlargement: Erythræmia, Hanot's cirrhosis, hæmochromatosis, Gaucher's type.

* Some included here are not primary blood diseases.

Enlargement of the Spleen, *continued.*

Enlargement in Children.—Especially in rickets, syphilis, and von Jaksch's anæmia; also in malaria and kala-azar.

Note.—Certain conditions with blood changes and splenomegaly are described among diseases of the spleen. It is very improbable that acholuric family jaundice and von Jaksch's anæmia are primarily diseases of the spleen. In 'splenic anæmia' the anæmia may result from abnormal conditions of the spleen, but it is possible that these are a sequence to some abnormality in the veins of the portal system—e.g., inflammation of the splenic vein.

III. MOVABLE SPLEEN.

Rare condition. Marked general enteroptosis usually present: but movable spleen is not found in more than 2 per cent of such cases. Rarely occurs alone. Mobility sometimes extreme. Usually some enlargement. Adhesions subsequently may fix organ.

Symptoms.—None associated specially with spleen; sometimes dragging pain. Very rarely: torsion of pedicle, with acute abdominal pain.

Treatment.—(1) Belt and pad. (2) Fixation by operation: satisfactory if other organs in place.

IV. RUPTURE OF SPLEEN.

Occurrence.—(1) Normal spleen; severe trauma necessary. (2) Malarial spleen: following a blow; *very rarely* spontaneously. Similarly in other enlargements. (3) Infarcts of spleen, very rarely.

Hæmorrhage, without rupture, may follow puncture of an enlarged spleen.

Symptoms.—(1) Sudden pain; followed by symptoms of (2) Internal hæmorrhage; and (3) Fluid in peritoneum.

Treatment.—Laparotomy, and removal of spleen.

V. INFARCTS, NEOPLASMS, AND CYSTS OF THE SPLEEN.

Infarcts.—Spleen is most common site next to kidneys.

ORIGIN.—(1) Emboli in splenic arteries: (a) Simple—endocarditis, cardiac thrombi; (b) Infective—infective endocarditis, sepsis. (2) Thrombus formation—e.g., in typhoid.

MORBID ANATOMY.—Either red or white infarcts. Usually multiple. Size, ¼ to 3 inches.

SYMPTOMS.—Pain and tenderness in left hypochondrium. Spleen may be palpable. Occasionally friction sound. Fever.

Tuberculosis.—Very common; secondary to infection elsewhere.

Neoplasms and Gummata.—Very rare.

Cysts.—Hydatid is most common. Others very rare.

VI. SPLENIC ANÆMIA.

(*Banti's Disease. Primary Splenomegaly with Anæmia.*)

A chronic disease of unknown origin, characterized by enlargement of the spleen, anæmia, recurrent hæmorrhages, and, in later stages of certain cases, cirrhosis of the liver.

Several conditions have been described as Banti's disease, splenic anæmia, primary splenomegaly: these are accepted now, provisionally, as one group. Banti's disease is applied to a type with a late cirrhosis of the liver, jaundice, and ascites. Gaucher's splenomegaly is not included. Splenomegaly in children and von Jaksch's type are probably of different origin.

Etiology.—

SEX.—Usually males.

AGE.—Onset often in early adult life or late childhood, especially Banti's group. Splenic anæmia without hepatic cirrhosis comes later.

Morbid Anatomy.—

1. SPLEEN.—Very large; firm, thick capsule; infarcts common: much fibrosis.
2. SPLENIC VEINS.—Phlebitis and obstruction almost invariably present. Portal vein may be similarly affected. Dilatation of œsophageal and other veins distal to obstruction.
3. LIVER.—In Banti's group, liver contracted, with atrophic interlobular cirrhosis. In other cases passive congestion common.

BONE-MARROW, LYMPHATIC GLANDS.—Normal.

Pathogenesis.—Uncertain. Principal theories:—

1. *Primary disease of spleen.*—Based on following arguments: (a) Splenomegaly is earliest symptom; (b) Splenectomy is a cure. Latter not conclusive, since splenectomy also cures splenomegaly secondary to thrombosis of portal vein.
2. *Disease or obstruction of splenic veins* primary, with secondary enlargement of spleen.—Based on arguments: (a) Phlebitis or obstruction of splenic or portal veins is almost invariably present; (b) Thrombosis of portal vein results in splenomegaly. The cause of the phlebitis is unknown.

The group may include several entities: thus, Banti's disease generally occurs at earlier age than cases without hepatic cirrhosis.

Symptoms.—Chronic; slowly progressive; often many years.

1. ENLARGEMENT OF SPLEEN.—To umbilicus or below; smooth and painless; size attracts attention.
2. ANÆMIA.—Slow advance; final extreme. In rare cases rapid.
3. BLOOD CHANGES.—(a) Leucocytes: Leucopenia marked (1000–3000 per c.mm.); relative lymphocytosis. (b) Erythrocytes: secondary anæmia. Abnormal red and white cells rare.

Splenic Anæmia—Symptoms, *continued.*

4. HÆMORRHAGES.—Hæmatemesis most common, from œsophageal veins; *recurs for years*, with irregular and prolonged intervals; often profuse; may be fatal.

In Banti's disease, the following symptoms develop subsequently :—

5. LIVER.—Enlarged. (In pure splenic anæmia, often reduced in size.)
6. ASCITES.—Recurrent.
7. JAUNDICE.—Rarely more than icteroid tint.

Course.—Very slow. May commence in late youth or young adult with splenomegaly and slight anæmia, slowly progressive. Then hæmorrhages, recurrent sometimes with intervals of years. Lastly (forming Banti's disease), cirrhosis of liver and ascites.

Death may occur from (1) Hæmorrhages; (2) Ascites, (3) Anæmia.

Diagnosis.—From :—

GASTRIC ULCER.

Various conditions with splenomegaly, especially :—

CIRRHOSIS OF THE LIVER with splenomegaly. Resemblance may be close.

1. ALCOHOLIC CIRRHOSIS.—With hæmatemesis, ascites, and occasionally enlarged spleen. History often distinguishes.
2. SYPHILITIC CIRRHOSIS.—Spleen often very large, and similar symptoms. Wassermann reaction positive; liver nodular, and other signs of syphilis.
3. HANOT'S HYPERTROPHIC CIRRHOSIS.

LEUKÆMIA (blood examination). LYMPHADENOMA (glandular enlargement).

PERNICIOUS ANÆMIA.—Spleen rarely more than palpable, anæmia of primary type.

TROPICAL SPLENOMEGALY, KALA-AZAR, MALARIA. (A condition resembling Banti's disease is common in Egypt: apparently not due to Leishmania.)

Rare diseases :—

ACHOLURIC FAMILY JAUNDICE.

GAUCHER'S PRIMARY ENDOTHELIOMA OF THE SPLEEN.

THROMBOSIS OR PHLEBITIS OF PORTAL OR SPLENIC VEINS.—Great enlargement of spleen; ascites may occur.

Treatment.—

MEDICAL.—Rest. Generous diet. Iron over prolonged periods.

SURGICAL.—Splenectomy. Results are improving, possibly as operation is performed earlier. Cure may occur. Considerable operative mortality, but this is diminishing.

VII. GAUCHER'S SPLENOMEGALY.

(*Primary Endothelioma of the Spleen.*)

Chronic splenomegaly of unknown origin, characterized histologically by the presence in the spleen of peculiar large cells, which are also found in other organs. Very rare: few cases recorded.

Etiology.—

SEX.—Females much commoner than males.

AGE.—Onset in early life. Possibly congenital. May be several in same generation.

Morbid Anatomy.—

SPLEEN.—Very large, gray-red colour.

HISTOLOGY.—Characterized by masses of peculiar large cells (20 by 40 μ) with very small round nuclei—atypical endothelial cells. Pigment containing iron is scattered in and between cells.

LIVER, BONE-MARROW, LYMPHATIC GLANDS.—Masses of similar cells.

Pathogenesis.—Uncertain whether condition is a *primary endotheliomata* with metastases, or whether cells are result of a stimulus causing hyperplasia.

Symptoms.—Very chronic: duration twenty years or more. *Health good.* Spleen greatly enlarged. Anæmia slight (some leucopenia). No gross hæmorrhages. No ascites or jaundice. Liver may be greatly enlarged in late stages.

Diagnosis.—Very rare disease. Diagnosis from splenic anæmia rarely possible; but note: (1) Females preponderate; (2) Early onset; (3) Chronicity and good health; (4) Anæmia slight; (5) No large hæmorrhages, jaundice, or ascites.

Treatment. None. Results of splenectomy at present unknown.

VIII. ACHOLURIC FAMILY JAUNDICE.

(*Congenital Family Cholæmia.*)

A rare hereditary condition characterized clinically by jaundice and enlarged spleen, and pathologically by abnormal fragility of the red cells.

General Characteristics.—

1. HEREDITARY.—Traced in several generations and affecting several members. Both sexes liable. Often noticed in childhood. *Sporadic* cases occur rarely, affecting several children but not previous generations.
2. SPLEEN enlarged.
3. JAUNDICE.—Generally slight. Bile present in serum, but usually *not in urine*, and stools not clay-coloured.
4. FRAGILITY OF RED CELLS.—Hæmolysed by sodium chloride 0·7 per cent. Normal cells resist 0·4 per cent. (Physiological 'normal saline' isotonic with blood is 0·85 per cent.)
5. BLOOD.—Ordinary secondary anæmia.
6. SYMPTOMS.—May be good health over long periods. Usually exacerbations of jaundice occur, with constipation, depression, and occasionally bile in urine.

Treatment.—Splenectomy: mortality low and results good. Unnecessary when health good.

(*See* note under SPLENOMEGALY, p. 742.)

IX. SPLENIC ENLARGEMENTS IN CHILDREN.

SPLENIC ANÆMIA OF CHILDREN.

(*Anæmia Infantum Pseudoleukæmia. Von Jaksch's Anæmia.*)

Etiology.—

AGE.—A few months and upwards.

PREDISPOSING FACTORS.—Syphilis and rickets definitely in some cases. In others both absent. Diarrhœa not uncommon. Occasionally, possibly, over-prolonged lactation.

(*See* note under SPLENOMEGALY, p. 742.)

Symptoms.—

1. SPLEEN AND LIVER ENLARGED.—Spleen often to umbilicus.
2. ANÆMIA.
3. BLOOD CHANGES.—(a) Erythrocytes: Extreme anæmia; 1,000,000 to 2,000,000 per c.mm.; hæmoglobin 10 to 40 per cent. (b) Leucocytes: Increased, 20,000 to 40,000 per c.mm. (c) Stained blood: Great variety of abnormal red and white cells; normoblasts, megaloblasts, often in large numbers; myelocytes, 10 to 25 per cent; may also be increase of lymphocytes. Occasionally: *Leucopenia*, with few abnormal leucocytes.

Progress.—Many die. Few recover completely. In others spleen remains enlarged while anæmia improves; may finally resemble splenic anæmia of adults.

Treatment.—General: especially hygiene and careful dieting. Iron over prolonged periods—e.g., ferri carbonas saccharatus. Splenectomy: high mortality.

RICKETS. CONGENITAL SYPHILIS.

May produce conditions identical with above. Some greater tendency to leucopenia with relative lymphocytosis.

CHAPTER CXXI.

DISEASES OF THE PITUITARY BODY.

I. MORPHOLOGY AND FUNCTIONS OF THE PITUITARY BODY.

Morphology.—Lies in the sella turcica, attached by the infundibulum to the base of the brain behind the optic chiasma. Weight in adults about 0·5 grm. Size: transverse diameter 12 to 15 mm.; vertical and antero-posterior diameters 5 to 8 mm. Divides at intraglandular cleft into two lobes.

1. ANTERIOR LOBE.—Envelops posterior lobe laterally and forms two-thirds of body.

HISTOLOGY.—Columns of epithelial cells with numerous blood-sinuses and blood-vessels. Three types of cells: (1) Chromophil, large cells containing granules: (a) eosinophil; (b) basophil, less numerous and mainly at periphery of lobe. (2) Chromophobe, small clear neutrophil cells. Probably types are same cell in various secretory stages—i.e., chromophobes have discharged their secretion, eosinophils have recently formed it, and basophils possibly are storing it.

2. POSTERIOR LOBE AND INFUNDIBULUM.—Consists of two parts, pars intermedia and pars nervosa.

HISTOLOGY.—

(a) *Pars Intermedia.*—A narrow lining on the posterior wall of the cleft consisting of polygonal neutrophil cells. 'Hyaline bodies' present, probably a secretion formed by degeneration of the cells. Also sometimes definite colloid (contains no iodine). Blood-vessels not numerous.

(b) *Pars Nervosa.*—Mainly neuroglia: no true nerve-cells or fibres, and no evidence of formation of secretion. 'Hyaline bodies' present, in passage from pars intermedia to cavity of 3rd ventricle: numerous after thyroidectomy. Blood-vessels scanty.

Development.—Two separate embryonic tissues contribute.

(1) A process develops from the cerebral vesicle, the thalamencephalon, finally forming the pars nervosa and infundibulum: In the latter a cavity connecting with the 3rd ventricle persists in certain animals. This unites with:—

(2) Rathke's pouch, an outgrowth from the primitive buccal cavity or stomodæum. Early in 4th week, the neck is constricted by growth of the sphenoid cartilage. The cavity persists as the intraglandular cleft, the posterior wall forming the pars intermedia, this portion of the posterior lobe thus having same origin as the anterior lobe.

Functions.—Evidence obtained by administration of gland, removal, etc.

ANTERIOR LOBE.—

1. ADMINISTRATION, by mouth or injection.—No definite effects.
2. EXPERIMENTAL REMOVAL.—Rapidly fatal if complete. Partial removal produces (i) genital infantilism, (ii) partial stunting of growth; adiposity common, but probably of posterior lobe origin.
3. CLINICAL OBSERVATIONS.—Over-activity: produces gigantism and acromegaly. Under-activity: possibly produces infantilism, type Lorain.

Summary.—Secretion is connected with: (i) Growth of skeleton, also of skin and soft tissues. (ii) Sexual organs, growth and activity. Possibly: (iii) Calcium and magnesium secretion; (iv) Carbohydrate metabolism.

POSTERIOR LOBE.—

1. ADMINISTRATION.—By mouth, little or no effect. Hypodermically, action important: (i) Blood-pressure raised

Pituitary Body—Functions, *continued.*

by constriction of vessels. (iii) Contraction of unstriped muscle, e g , uterus, bladder, intestines. (iv) Secretion of urine diminished. (v) Glycosuria and hyperglycæmia. (v) Galactagogue action: transient: daily amount not increased.

Notes. (1) A depressor substance is present in extracts, causing a preliminary fall in blood-pressure. Mainly produced during manufacture. (2) A second injection shortly after first does not produce the specific effects, but the depressor action occurs hence interval should be at least 3 hours. (3) Secretion of urine. In ether-anæsthetized animals, injections dilate the kidney and its vessels and increase the urine, but in unanæsthetized animals and in human beings, normal or with diabetes insipidus the secretion is halved (Kennaway and Mottram). (4) Glycosuria. Only occurs until glycogen is exhausted protein and fats are not converted into carbohydrate (5) Pars nervosa and intermedia have same action, but former is more powerful

Other effects of administration Heart slowed and beats more powerful not constant, and injections are dangerous in cardiac weakness owing to depressor action. Contraction of bronchial muscles Diminished range of respiration. Diminished pancreatic secretion

2 EXPERIMENTAL REMOVAL, PARTIAL OR COMPLETE —No obvious effects.

3. CLINICAL OBSERVATIONS —Underactivity produces diabetes insipidus.

WHOLE GLAND.—Total removal is fatal Certain lesions, also experimental ligature of stalk and almost complete removal (some anterior lobe remaining), produce infantilism, stunting of growth, and *adiposity*, origin of last doubtful

SUMMARY.—Pituitary has a control upon :—

1. Growth of skeleton , also of soft tissues.
2. Sexual organs : growth and functions.
3. Adiposity
4. Secretion of urine
5. Various functions including carbohydrate metabolism, contraction of unstriped muscle, blood pressure

II. SYMPTOMATOLOGY OF DISEASES OF THE PITUITARY BODY.*

A. **Neighbourhood Symptoms.**—Due to local pressure-effects . occur with tumours or hyperplasia.

1. HEADACHE.—Bitemporal. Possibly distention of capsule.
2. OCULAR MANIFESTATIONS.—From pressure on optic chiasma and tracts.

*Classification based on Cushing.

(a) PRIMARY OPTIC ATROPHY.—Not uncommon, usually unilateral: 'choked disc' may occur on other side.

(b) ALTERATIONS IN THE VISUAL FIELDS.—Usually asymmetrical. May commence as:—

(i) *Bitemporal hemianopia.*—Classical type, but not very frequent. Begins in upper margins.

(ii) *Loss of colour vision* (red initially).—Often very early: may begin as central scotoma, or peripherally, or both together.

(iii) *Central scotoma.*—Extends to form temporal hemianopia.

(iv) *Unilateral blindness.*—Opposite side subsequently.

(v) *Homonymous hemianopia.*—Occurs occasionally.

Blindness finally may be complete.

(c) OCULAR PALSIES.—Not common. From pressure on 3rd, 4th, and 6th nerves in cavernous sinus.

3. CHANGES IN SELLA TURCICA.—(a) Thickening of clinoid processes. Only as part of bony overgrowth in acromegaly and gigantism. (b) Thinning of clinoid processes and walls of sella. With adenomata. (c) Destruction of outlines. Usually malignant tumours. (d) Sella abnormally small. Primary hypoplasia. (e) Sella normal. In tumours of pituitary stalk.

4. PRESSURE ON THE BRAIN.—Sites: (a) Hippocampus gyrus, producing uncinate fits: not uncommon. (b) Frontal lobes, rarely: psychical changes.

5. NASOPHARYNGEAL SYMPTOMS.—Epistaxis, discharge of mucus or of cerebrospinal fluid. Occasionally, in malignant tumours.

Rarely: Exophthalmos: from pressure on cavernous sinus. Trigeminal neuralgia. Distention of veins of scalp and eyelid.

B. **General Pressure Effects.**—From rise of intracranial pressure. Rarely marked, except headache. Vomiting and choked disc are both unusual.

C. **Secretory or Glandular Symptoms Proper.**—In conditions due to dyspituitarism, i.e., pathological action of the pituitary, the recognition of the exact variation is often uncertain and is complicated by the following factors: (1) Either lobe may be over- or underactive, and overactivity of one may be associated with underactivity of the other; (2) Overactivity may be followed by underactivity; (3) Effects vary with age at onset.

PROVISIONAL SUMMARY OF CLINICAL SYNDROMES.—

ANTERIOR LOBE.—

1. *Oversecretion.*—(1) Before union of epiphyses: gigantism. (2) After union of epiphyses: acromegaly.

2. *Undersecretion.*—(1) Before puberty: infantilism, type Lorain. (2) After puberty: not recognized.

POSTERIOR LOBE.—

1. *Oversecretion.*—Not recognized: possibly certain forms of glycosuria and hyperglycæmia.

2. *Undersecretion.*—Diabetes insipidus.

Symptomatology of Diseases of the Pituitary Body, *continued.*

WHOLE GLAND.—

Undersecretion.—Syndromes of adiposity, genital dystrophy or atrophy, stunting of growth and infantilism, varying in type principally according to age at onset: (*a*) Before puberty: 'pudding-face type' (Fearnsides). (*b*) During puberty: dystrophia adiposo-genitalis (Fröhlich). (*c*) After puberty: eunuchoidal types.

Polyglandular Syndromes.—Clinical condition sometimes suggests that other ductless glands are also at fault, but knowledge very uncertain. Note relations to: (1) *Sexual organs*: Pituitary enlarges in pregnancy and after castration. In eunuchs, skeletal overgrowth and adiposity occur. (2) *Thyroid gland*: After thyroidectomy, the pituitary enlarges and histologically appears very active: also in myxœdema, cretinism, and parenchymatous goitre. In exophthalmic goitre, pituitary is inactive.

III. ACROMEGALY.

A condition characterized clinically by excessive growth of certain portions of the skeleton, especially exhibited in the face and extremities, by overgrowth of the soft tissues, and, pathologically, by hypersecretion of the pituitary.

Note.—Hyperpituitarism of the anterior lobe produces skeletal overgrowth, resulting in: (1) Gigantism, if occurring previous to ossification of epiphyses. (2) Acromegaly, if subsequent to such ossification: in the latter event, the skeletal overgrowth is in the acral portions, i.e., periosteal, though not entirely, since the lower jaw increases in length. A giant may become acromegalic, since the hypersecretion may subside and then recur, ossification of epiphyses occurring in the interval. Minor grades of this hyperpituitarism are probably common.

Two stages often recognizable, hyposecretion and its phenomena superseding the hypersecretion: especially exhibited by (1) carbohydrate metabolism, (2) sexual powers, (3) physical strength.

Etiology.—

AGE AT ONSET.—Commonest in third decade.

SEX.—Females 60 per cent.

PREDISPOSING CAUSES.—Unknown. Hereditary factor.

Pathology.—

1. PITUITARY BODY.—Always evidence of hypersecretion, present or past, of anterior lobe: (1) *Adenoma*, simple, fibrous, or malignant; never sarcoma. (2) *Struma*, viz., numerous active eosinophil or large chromophobe cells.
2. SKELETAL CHANGES.—Overgrowth of bone at bony prominences and sites of stress, e.g., origin and insertion of muscles, 'the osteoblasts being hypersensitive to ordinary stresses' (Keith).

3. SOFT TISSUES.—Thickening of skin and subcutaneous tissues (principal cause of large hands and feet).

OTHER DUCTLESS GLANDS.—Thyroid rarely normal: atrophic, enlarged, or goitrous. Various changes described in other glands.

Symptoms.—

COMMON EARLY COMPLAINTS.—(a) Increase of size in features, hats, gloves, and boots; (b) Visual changes; (c) Headache.

The varied symptoms described above under SYMPTOMATOLOGY may occur. Note especially hemianopia and the ocular manifestations. General pressure-effects rare. May be previous gigantism.

SYMPTOMS DUE TO ALTERATIONS OF SECRETION.—

a. SKELETAL CHANGES AND INCREASE IN SIZE.—

Face elongated and enlarged.—Especially lower jaw: teeth may protrude 1 inch beyond upper. Also superior maxilla, zygoma, and other prominences. Head in general enlarged.

Hands and feet enlarged.—Uniformly (from subcutaneous tissue increase and exostoses at tendon attachments). Arms rarely affected. Fingers not 'clubbed'.

Soft tissues increased.—Nose, eyelids, tongue, and especially *ears*. Skin thick.

Kyphosis and lordosis. *Thorax* increased in size: respiratory expansion slight. Sternum and clavicles enlarged.

Use of limbs not affected by changes.

b. CARBOHYDRATE METABOLISM.—Early stage: glycosuria and hyperglycæmia. Later stage: hypoglycæmia and high carbohydrate tolerance.

VARIOUS AND OCCASIONAL SYMPTOMS.—(1) Early stage: (a) Libido; (b) Physical strength. (2) Late Stage: (a) Impotence or amenorrhœa; (b) Physical and cardiac weakness.

POLYGLANDULAR SYNDROMES.—Goitre and changes in other ductless glands: e.g., combined myxœdema and acromegaly occur occasionally.

RADIOGRAPHS.—Note especially —

1. SELLA TURCICA.—May be: (a) Thickening of clinoid processes: a part of the general bony overgrowth, (b) Thinning of clinoid processes and enlargement of sella. Sella is never under the average dimensions.
2. HANDS AND FEET.—The terminal phalanges have characteristic 'tufts'.

Diagnosis.—Difficult in early stages. *X-ray* of sella turcica and cranium of great value (when perfect). Occasional diagnosis from —

MYXŒDEMA.—Bones unaffected. Dry hair and skin, etc. Effects of thyroid therapy.

OSTEITIS DEFORMANS.—Late age. Tibiæ curved. Lower jaw and soft tissues unaffected.

ARTHRITIS DEFORMANS.—Head not enlarged. Pain. Limited movements.

Acromegaly—Diagnosis, *continued.*

PULMONARY OSTEO-ARTHROPATHY — Fingers clubbed Heart or lung affections.

Course and Prognosis.—Chronic forms. 10 to 50 years. Acute forms (malignant tumours) · 6 years or less.

TERMINATION —From cerebral tumour, cardiac weakness, very rarely, diabetic coma

Notes on Gigantism.—Anthropologists consider persons over 6 feet 8 inches as 'giants'. Giants are: (1) Pathological—i e, hyperpituitarism (40 per cent of all giants). (2) Normal; appearance often suggests acromegaly, usually weak, physically and mentally, and die young, 20 to 30 years.

Treatment.—

ORGANOTHERAPY —At present unsatisfactory contra indicated in early stages with 'hyperpituitarism' and glycosuria.

OPERATION — For relief of intracranial pressure and to save sight

IV. DYSTROPHIA ADIPOSO-GENITALIS (Frohlich).

A condition due to deficient secretion of the pituitary gland (hypopituitarism), and characterized by obesity and arrest of development of sexual characteristics.

Hypopituitarism and Adiposity. — In certain forms of hypopituitarism a group of symptoms occur, varying with age at onset, but characterized in general by (1) skeletal undergrowth, (2) genital dystrophy or atrophy, (3) adiposity Deficient growth and sexual changes can be safely ascribed to the anterior lobe Adiposity is usually ascribed to the posterior lobe, but does not follow its complete removal The entire syndrome is reproduced experimentally only by (a) Ligature or division of the infundibulum (i e, whole gland affected); (b) Removal of posterior and most of anterior lobe (Cushing)

Morbid Anatomy (1) Often a tumour of pituitary body, e g, sarcoma, gumma. Occasionally (2) Primary hypoplasia of gland, (3) Serious effects on gland or the paths of its secretion produced by extrinsic tumours, injuries, or internal hydrocephalus, e g, from previous meningitis, cerebellar tumours.

Types.—Following can be correlated (approximately) to age of onset.—

1. ONSET BEFORE PUBERTY. — Fearnsides' 'pudding-face type'. Adiposity enormous and universal. Stature often not less and may be greater than normal. Genital dystrophy not marked, but usually early death from tumour.
2. ONSET DURING PUBERTY.—Terms 'dystrophia adiposo-genitalis', and 'Fröhlich's disease' are usually confined to this group.

(a) DEFICIENT GROWTH OF SKELETON.—Insufficient to constitute 'dwarfism'. In males, tendency to feminine (or neutral) outlines—viz., broad pelvis, genu valgum common, delicate tapering of fingers and fine extremities.

(b) SEXUAL INFANTILISM.—Sexual organs remain infantile, and secondary sexual characteristics absent—i.e., no hair on face, pubes, or axilla, mammæ small, no menstruation or spermatozoa.

(c) ADIPOSITY.—General, but specially in feminine sites—e.g., over hips.

3. ONSET AFTER PUBERTY.—

a. EUNUCHOIDAL TYPE.—(i) Atrophy of sexual organs, impotence or amenorrhœa. (ii) Adiposity in males, specially in feminine sites.

b. ADIPOSIS DOLOROSA.—*See* p. 335. Not invariably of pituitary origin.

c. MILD GRADES.—Not infrequent. Obesity, impotence, or amenorrhœa.

Notes on Symptoms.—

1. VARIOUS OTHER SYMPTOMS OF DYSPITUITARISM.—(a) Carbohydrate metabolism: hypoglycæmia and high tolerance. (b) Skin smooth: may be pigmentation. (c) Somnolence. (d) Low temperature and blood pressure: may be polyuria.

2. NEIGHBOURHOOD SYMPTOMS.—Occur with tumours (*see* SYMPTOMATOLOGY OF DISEASES OF THE PITUITARY BODY, p. 748).

Treatment.—Operation on tumour to relieve pressure. Pituitary extracts to be tried: dosage judged by watching sugar tolerance.

V. TUMOURS OF THE PITUITARY BODY.

Pathology.—Size rarely exceeds a walnut.

1. PITUITARY TUMOURS.—

a. ADENOMA.—Cushing's 'chromophobe struma'. Varies from simple hyperplasia to apparent malignancy (but never metastases, structures not invaded, and no mitotic figures). Is sole type occurring in hyperpituitarism. Formerly often regarded as sarcoma.

b. SARCOMA. CARCINOMA.

2. EXTRAPITUITARY TUMOURS.—Very rare.

a. Infundibular tumours. Teratoma commonest: also cysts, endothelioma.

b. From pituitary rests, especially neck of Rathke's pouch: adenoma, epithelioma.

c. From neighbouring structures.

Symptoms.—*See* SYMPTOMATOLOGY OF DISEASES OF THE PITUITARY BODY, p. 748.

CHAPTER CXXII.

INFANTILISM.

Retention, in varying degrees, of the characteristics of childhood—sexual, bodily, and mental

Classification.—

1 ALTERATIONS OF INTERNAL SECRETIONS
   (a) THYROID GLAND—Cretinism.
   (b) PITUITARY GLAND—Types (i) Pudding face type (before puberty) (ii) Frohlich's 'dystrophia adiposo genitalis' (about puberty), (iii) Type Lorain or ateleiosis Possibly (iv) With diabetes insipidus
   Possibly belonging to this group —
   c INTESTINAL INFANTILISM—With large, loose, fatty stools Includes 'coeliac' and 'pancreatic' infantilism. (See COELIAC DISEASE)
   d RENAL INFANTILISM.—See p 350
   e PROGERIA—Possibly of suprarenal origin
   f SEXUAL GLANDS
2. CACHECTIC CONDITIONS—Any chronic disease in childhood may delay development producing partial infantilism, e g, (a) Syphilis, usually congenital (b) Heart disease congenital or acquired (c) Malaria ankylostomiasis hookworm disease (d) Nervous and mental diseases many types e g microcephaly possibly Mongols (e) Early alcoholism possibly also tobacco

Notes.—

1 Minor degrees of infantilism probably not infrequent Many cases are unclassifiable Vitamin deficiency may be a cause also general underfeeding Probably other causes
2 BRISSAUD'S TYPE—Was ascribed to hypothyroidism but description agrees with early type of Frohlich's disease
3. TYPE LORAIN OR ATELEIOSIS—Proportions of a miniature adult No facial, pubic or axillary hair Sexual organs small Voice high pitched Intelligence varies usually normal Also called 'anangioplastic infantilism' from theory of vascular origin Probably deficiency of pituitary anterior lobe
4 PROGERIA (Hastings Gilford)—Very rare Premature senility with *extreme fibrosis* especially of arteries and kidneys Dry skin, loss of hair, and appearance of old age Death before puberty
5 MONGOLS—Origin unknown Often at end of large families Mongoloid facies, short head, protruding tongue Mentally defective. Physical defects common, e g, heart, strabismus, nystagmus

PRECOCITY occurs with certain tumours of suprarenal possibly also of pineal gland

'DWARFISM' is an extreme deficiency of stature, but is not necessarily infantilism — e g, spinal caries, severe rickets, or achondroplasia.

Section XI.—DISEASES OF THE NERVOUS SYSTEM.

CHAPTER CXXIII

SENSORY AND MOTOR PATHS.

I. PATHS OF SENSATION AND AFFERENT TRACTS.

1 **In the Peripheral Nerves.**—

VARIETIES OF SENSORY IMPULSES Head Sherren and Rivers have shown that three systems of sensory impulses exist, each with its own fibres

a DEEP SENSIBILITY Fibres convey sensations from muscles, joints, and bones and ascend consecutively in muscle, tendon and *motor* nerves Functions (i) Deep pressure and pain (ii) Sense of passive position—muscle sense, (iii) Accurate localization of pressure

b PROTOPATHIC SENSATION Fibres in sensory nerve Functions (i) Pain (pin prick), (ii) Extremes of heat and cold – viz, below 20° C and above 40° C Sensation (e g, pin-prick) radiates widely and power of localization is very slight Areas supplied overlap considerably

c EPICRITIC SENSATION—Fibres in sensory nerves Functions (i) Light touch (ii) Temperature between 20 and 40° C, (iii) Localization—very accurate. Also tactile discrimination, e g compass points, agnosthesia, e g appreciation of differences in size of objects No overlapping of areas supplied

Relations between areas of epicritic and protopathic loss vary with site of lesion (i) In peripheral nerve, epicritic loss greater than protopathic, and difference increases as periphery is approached (ii) In plexus, areas equal (iii) In dorsal root epicritic less extensive than protopathic. *Deep sensibility* is retained unless tendon, are divided.

SENSORY CHANGES OCCURRING IN NERVE LESIONS—

a. PAIN—Varies with cause and severity of lesion If slow compression, slight or nil, in inflammation, very severe.

b ANÆSTHESIA.—Varies with site and cause of lesion.

c PARÆSTHESIA (numbness, tingling, etc).—May occur with no anæsthesia.

d. HYPERALGESIA—Especially in areas where epicritic sensation is lost and protopathic is present.

Objective sensory changes are generally less marked than motor changes

Paths of Sensation and Afferent Tracts, *continued.*

2. **In the Spinal Cord.**—On entering the spinal cord, the sensory fibres are immediately rearranged and subsequently ascend in three fresh groups (researches by Head and Thompson).

GROUPS OF SENSORY FIBRES IN THE SPINAL CORD.—

1. TACTILE IMPULSES.—Include: (a) Light touch (epicritic); (b) Deep touch (deep sensibility).
2. IMPULSES OF PAIN AND TEMPERATURE.—Include: (a) All sensations of heat and cold, both epicritic and protopathic; (b) Pain, viz. (i) pin-prick (protopathic), (ii) bone pain (deep sensibility).
3. IMPULSES OF LOCALITY, PASSIVE POSITION, ETC.—Include: (a) Localization of touch; (b) Tactile discrimination; (c) Acuæsthesia; (d) Sense of passive position; (e) Sense of muscle movement, or muscle sense; (f) Vibration.

All primary sensory neurons end about cells on the same side of the cord.

PATH OF THE THREE GROUPS IN THE SPINAL CORD.—

1. TACTILE IMPULSES.—(a) The primary sensory neurons at once enter the *posterior columns*, and ascend in these almost the entire length of the cord. At various levels, the fibre gives off collaterals which enter the posterior horns and end around cells. (b) The fibres from these cells—i.e., secondary sensory neurons—immediately cross the cord in the posterior commissure, and pass to a spino-thalamic tract in the *anterior ground bundles* (of the opposite side), forming an ascending tract between the direct pyramidal tract and the anterior horn and root. Owing to this long course in the posterior columns and numerous collaterals, light touch is rarely completely abolished in lesions of the cord.

   Termination of this Spino-thalamic Tract.—In the medulla, the fibres are joined by other sensory fibres, e.g., from cuneate and gracilis nuclei, and later also by sensory fibres from the head. Combined tract forms the *median* (or mesial) *fillet* and ascends to optic thalamus.

2. IMPULSES OF PAIN AND TEMPERATURE.—The primary sensory neurons enter the *posterior horns*, and shortly end about cells. The secondary sensory neurons from these cross the cord at once in the posterior commissure close to the central canal, and ascend in a spino-thalamic tract close to Gowers' antero-lateral ascending tract (of the opposite side). Syringomyelia injures the fibres while *crossing the cord*; being arranged here in order of heat, cold, pain, from before backwards, heat may be affected more than cold, and cold than pain.

   Termination of this Spino-thalamic Tract.—Joins the median fillet and ascends to optic thalamus.

3. IMPULSES OF LOCALITY, PASSIVE POSITION, ETC.—The primary sensory neurons at once enter the *posterior columns*, and ascend on the same side to the cuneate and gracilis

nuclei. Collaterals from certain of the fibres end round cells in Clarke's column, whence the secondary sensory neurons pass outwards through the posterior horn and lateral pyramidal tract to the direct and ventral cerebellar tracts, and ascend in these on same side as entry. This group of impulses is affected in tabes.

Termination of Tracts.—Three tracts are formed :—

i. *Fibres in posterior columns* ascend to and end in cuneate and gracilis nuclei. Relay fibres decussate in fillet, join median fillet of opposite side, and ascend to optic thalamus.

ii. *Direct cerebellar or dorsal spino-cerebellar tract* ascends through inferior cerebellar peduncle to vermis.

iii. *Ventral cerebellar or Gowers' antero-lateral ascending tract* passes through medulla, pons, and superior peduncles to cerebellum. Certain fibres run to corpora quadrigemina.

The last two tracts convey impulses from muscles and joints to the cerebellum and are concerned in maintenance of equilibrium.

3. **In the Brain.**—There are two centres for conscious appreciation of sensations, viz., in optic thalamus and in cerebral cortex. All afferent fibres end in the optic thalamus.

OPTIC THALAMUS.—

1. A mass of gray matter. Is a centre for conscious appreciation of certain crude sensations, especially of pleasure and discomfort, including pain, temperature, touch, and consciousness of changes of such states.

Cerebral cortex can control activities of this centre.

2. Other afferent fibres terminate here, and relays, conveying finer sensations, commence. They pass through posterior limb of internal capsule and corona radiata to cerebral cortex, where they reach consciousness.

CEREBRAL CORTEX.—Such impulses ascending to cortex are redistributed and collected into five main groups (Head and Holmes) : (i) Recognition of position of limbs and body in space and of passive movements ; (ii) Recognition of finer tactile differences and intensity of touch ; (iii) Recognition of size, weight, shape, and texture of objects ; (iv) Localization of a stimulated spot ; (v) Sensations of temperature. They are localized in the cortex in order from the Rolandic fissure back to the supramarginal and angular gyri.

II. MOTOR TRACTS.

Voluntary motor impulses commence in cerebral cortex, and on path to muscles pass through at least two neurons, i.e., upper and lower motor neurons. The upper motor neurons cross the mid-line.

Upper Motor Neurons.—

1. CEREBRAL CORTEX.—Fibres commence from large pyramidal (Betz) cells in motor cortex anterior to fissure of Rolando.

Motor Tracts, *continued.*

Centres *from above downwards* are arranged in order leg, trunk, arm, face. Or, in greater detail anus, toes, ankle, knee, hip, abdomen, chest, shoulder, elbow, wrist, hand, thumb, neck, eyelids, ear, nose, mouth, and tongue.

2 INTERNAL CAPSULE —The fibres, forming the pyramidal tracts, constitute and pass through the corona radiata to internal capsule, where they are arranged *from before backwards* in order (i) In front of angle (eyes and head) (ii) At angle tongue, mouth. (iii) Anterior two-thirds of posterior limb shoulder, elbow, wrist, fingers, thumb, trunk hip, knee, ankle, toes (In posterior third of posterior limb are sensory fibres ascending from optic thalamus to sensory cortex, and posterior to these are the optic radiations)

3. CRUS, PONS, AND MEDULLA In the crus pyramidal tract occupies middle two-fifths of crusta From here to medulla certain fibres leave the tract, cross the mid line and terminate in nuclei of motor cranial nerves of opposite side In the medulla, the pyramidal tracts form the anterior pyramids Here most fibres cross mid line forming the decussation of the pyramids, and descend as the lateral or crossed pyramidal tract of the cord Remaining fibres continue uncrossed as the direct pyramidal tracts.

4 SPINAL CORD —Two tracts are present —

i LATERAL or CROSSED PYRAMIDAL TRACT Situated in lateral columns Fibres end about motor cells of anterior horns

ii DIRECT PYRAMIDAL TRACT (Turck's) —In anterior columns Fibres cross, and end in anterior horn of opposite side Crossing is complete at mid thoracic region

Lower Motor Neurons.—Fibres commence from motor cells of anterior horns, and pass through anterior roots and peripheral nerves to end-organs in the muscles In the brain they commence from the cranial nuclei in pons and medulla

Subsidiary Descending Tracts. The course and functions of these are less known Most definite are —

1. RUBRO-SPINAL TRACT (Bundle of Monakow) —

ORIGIN —From dentate nucleus (cerebellum) fibres cross to opposite red nucleus, in tegmentum of crus Rubro-spinal tract commences from red nucleus immediately decussates with opposite tract, and crosses mid-line Communications are effected with motor cranial nuclei In the cord, tract is situated in lateral columns, anterior to crossed pyramidal tract, and fibres end in connection with anterior horn cells

FUNCTION.—Main efferent cerebellar tract: connects cerebellum with same side of body (doubly crossed). Concerned in maintenance of equilibrium and co-ordination of antagonistic muscles.

In conjunction with this tract are —

Lenticulo-rubro-spinal Tract. — Fibres from lenticular

nucleus to red nucleus of same side · thus connects with opposite side of body. Lesions produce tremors resembling paralysis agitans and rigidity of special type.

ii *Thalamo spinal Tract* —Fibres from optic thalamus

2. TECTO-SPINAL TRACT —

ORIGIN.—From corpora quadrigemina, partial decussation In cord, situated in antero lateral columns, anterior to rubrospinal tract

FUNCTION —Concerned in reflex actions dependent on auditory and visual stimuli (e g, hitting a fast moving ball)

3 VESTIBULO-SPINAL TRACT (Antero lateral descending tract) —

ORIGIN —From Deiters nucleus in the pons. Tract is uncrossed In cord is situated in anterior columns, and fibres terminate about anterior horn cells

FUNCTIONS —Connects cerebellum with same side of body Is concerned in tone of muscles and in position of body, especially in correlation with vestibular and ocular stimuli

Deiters nucleus (lateral nucleus of eighth nerve) has connections with (i) Semicircular canals, through vestibular nerve, (ii) Cerebellum roof nuclei through middle peduncle, (iii) Motor nerves of eye, through posterior longitudinal bundle

Other small tracts in cord also show descending degeneration —

TRACT OF MARIE In anterior columns is continuation of posterior longitudinal bundle

OLIVO SPINAL TRACT (Bundle of Helwig) - -From optic thalamus through inferior olive Close to vestibulo spinal tract

COMMA TRACT —Between columns of Goll and Burdach Consists mainly of descending branches of afferent fibres

SEPTO MARGINAL BUNDLE —Adjoining posterior longitudinal fissure mainly proprio spinal fibres (The last two tracts do not degenerate in tabes)

Lesions of the Motor Tracts. Destructive lesions of motor tracts result in paralysis of muscles supplied Impulse from brain in upper neuron normally inhibit activities of lower neuron, which are increased in their absence Nutrition of lower neuron is independent of upper neuron, and its involuntary activities can continue when isolated Two types of paralysis thus occur.

		Upper Neuron Lesion	*Lower Neuron Lesion*
1	Paralysis	Spastic	Flaccid
2	Wasting	From disuse only	Rapid and marked
3	Deep reflexes	Increased, e g, knee jerks, Babinski extensor reflex	Absent
4	Electrical reactions	Unchanged	Reaction of degeneration, partial or complete
5.	Sensory and trophic changes	Absent	Present, degree varies
6.	Contractures	Mainly of spastic muscles	Mainly of unantagonized muscles

Motor Tracts, *continued.*

Brown-Séquard's Syndrome.—Occurs in unilateral transverse cord lesions.

1. ON SIDE OF LESION.—
   a. IN AFFECTED SEGMENT.—(i) Zone of anæsthesia, with area of hyperæsthesia above; (ii) Atrophic (lower motor neuron) paralysis.
   b. BELOW AFFECTED SEGMENT.—(i) *Paralysis*, of upper motor neuron type. (ii) Sensory changes: Loss of sense of position, of localization of touch, and of tactile discrimination, e.g., size and shape of objects (stereognosis) and compass points, and also vibrations of tuning-fork.
2. ON OPPOSITE SIDE TO LESION.—Loss of sensation of pain and temperature. Touch may be slightly affected. No motor changes.

III. REFLEXES.

A reflex is a muscular contraction in response to an afferent, sensory, stimulus. Its occurrence involves the integrity of a 'reflex arc' including an afferent nerve, an efferent nerve (lower motor neuron), a connection between the two in the central nervous system, and a muscle capable of contraction.

A reflex may vary from normal in being: (1) *Diminished or absent*: from interruption at any point of arc. (2) *Increased*: impulses in upper motor neuron (pyramidal tracts) inhibit activity of lower motor neuron, and deep reflexes become increased if such impulses are interrupted. (3) Altered in type, e.g., Babinski's sign.

1. **Deep or Tendon Reflexes.**—Principal are: (i) *Knee-jerk*: segments of cord involved are second, third, and fourth lumbar. (ii) *Tendo Achillis*: first sacral segment. Also: jaw-jerk (fifth nerve), supinator (fifth cervical), triceps (sixth and seventh cervical).

2. **Superficial Reflexes.**—Principal are: (i) *Plantar reflex*: first, second, and third sacral segments. (ii) *Abdominal*: eighth to twelfth dorsal. Conjunctival, palate, cremasteric, and others.

   Superficial abdominal reflexes are diminished in lesions of pyramidal tracts (cf. deep reflexes), e.g., in cerebral lesions. All superficial reflexes depend considerably on condition of skin—e.g., with cold, damp, or laxness, are often absent; should be tested for repeatedly, and absence must be cautiously interpreted.

Knee-jerks.—

A. INCREASED.—Cause may be: (1) Functional, e.g., hysteria, neuroses. (2) Organic, viz., interference with conduction of impulses in upper motor neuron, e.g.: (*a*) In brain: hemiplegia, dementia paralytica. (*b*) In cord: lateral sclerosis, disseminated sclerosis, transverse myelitis.

B. ABSENT.—Cause: interruption of reflex arc, due to: (1) Muscular weakness: e.g., muscular dystrophy. (2) Lesions of afferent or efferent nerves: e.g., peripheral neuritis, trauma. (3) Lesions of lumbar segments of cord, e.g.: (*a*) Posterior roots, tabes; (*b*) Anterior horns, poliomyelitis.

If response be slight or doubtful, test with Jendrassik's reinforcement method.

Ankle-clonus.—Often occurs with lesions of pyramidal tracts: when present is evidence of organic lesion. 'Spurious clonus' may be organic or functional.

Plantar Reflex: Babinski's Sign—Stimulation of the sole in infancy produces upward movement of great toe, i.e., dorsiflexion, or 'extensor response'. After learning to walk, response becomes 'flexor': connected with action of great toe in walking. If the pyramidal tracts be interrupted, reflex again becomes 'extensor', i.e., a positive Babinski's sign.

Positive Babinski's Sign is definite evidence of organic lesion of upper motor neuron.

CHAPTER CXXIV.

DISEASES OF THE PERIPHERAL NERVES.

I. NEURITIS.

Causes.—Lesions of single peripheral nerves occur from: (1) *Trauma*: compression or division. Usual cause. (2) Extension of inflammation—e.g. from caries of bone. (3) Cold.

Morbid Anatomy.—*Interstitial* neuritis—i.e., inflammation of supporting fibrous tissue; later, nerve fibres are destroyed by pressure.

Symptoms.—With complete interruption of a mixed nerve, complete loss of motor, sensory, and trophic functions results. With partial interruption—e.g., by compression—sensory fibres suffer less than motor: there may be no sensory change, or slight epicritic loss.

1. MOTOR SYMPTOMS.—Of lower motor neuron type. *See* p. 759.
2. SENSORY SYMPTOMS.—*See* p. 755.
3. TROPHIC CHANGES.—*Skin*, in chronic conditions, dry, glossy, and tightly tretched; no sweating; often hairless; liable to ulceration. *Nails* brittle and furrowed; growth slow (from diminished circulation). *Bones* may be more fragile. *Joints*: occasionally effusion, thickening, and rarely ankylosis.

Prognosis.—

SLIGHT LESIONS (contusions or compression).—Recovery in a few days or weeks.

Neuritis—Prognosis, *continued.*

NERVE DIVIDED AND SUTURED.—Progress usually:—

1. PROTOPATHIC SENSATION.—Commences in 2 to 3 months, complete in 6 months.
2. EPICRITIC SENSATION.—Commences in 6 months, complete in 1 year or more.
3. MOTOR FUNCTIONS.—In upper extremity, division near wrist: recovery in 1 year. Near elbow and in plexus at least 2 years (Sherren).

Diagnosis.—Note:—

HYSTERICAL CONTRACTURES.—(1) All sensations affected over similar area, (2) 'Stocking' or 'glove' distribution; (3) No reaction of degeneration, (4) Flexors and extensors both affected (*See* HYSTERIA).

VOLKMANN'S ISCHÆMIC CONTRACTURE from tight bandages or splint.—(1) No anæsthesia, (2) No reaction of degeneration.

Treatment.—

OF DIVIDED NERVES.—Suture.

OTHER CAUSES AND GENERAL TREATMENT.—*See* MULTIPLE NEURITIS.

II. MULTIPLE NEURITIS.

(*Peripheral Neuritis. Polyneuritis.*)

Inflammation and degeneration of multiple peripheral nerves, resulting in disturbance of motor and sensory functions usually affecting the limbs, and of symmetrical distribution.

Etiology.—

1. TOXIC.—*Alcohol, lead, arsenic,* and mercury. Rarely from other metals and organic substances.
2. INFECTIOUS FEVERS, especially *diphtheria.* Rarely enteric, influenza, and others.
3. DIABETES.

Occasionally:—

4. Malaria, gout, and possibly rheumatism. Rarely syphilis.
5. Cachectic conditions: cancer, anæmia, etc. Rare.
6. No obvious cause: cold, over exertion.

Also dominating lesion in:—

7. *Beri-beri.*
8. *Leprosy.*

Morbid Anatomy.—Mainly *parenchymatous* neuritis. Wallerian degeneration of nerve fibres; interstitial changes slight.

Symptoms.—A general description of symptoms is given here, followed by an account of certain special forms, especially the alcoholic. Various causes have a selective action on special nerves. *See also* p. 764.

DISTRIBUTION.—Symmetrical. Distal portions of extremities nearly always first affected. Cranial nerves and those supplying trunk muscles rarely affected, except in special forms.

MOTOR SYMPTOMS.—*Loss of power*, especially in extensor muscles below knee and elbow, whence wrist-drop and foot-drop. Characteristics of lower motor neuron lesion present, viz.: (1) *Flaccid paralysis and wasting of muscles*; (2) Loss of deep reflexes; (3) Reaction of degeneration.

SENSORY SYMPTOMS.—Often *precede* motor symptoms. Tingling, numbness, hyperæsthesia, anæsthesia; extent of alteration and impairment of sensation very variable. *Tenderness of muscles.*

REFLEXES.—Deep reflexes lost. In lower extremities: *knee-jerks absent*, no ankle-clonus, plantar reflex flexor. (Occasionally in early stages of nerve irritation knee-jerks are increased, but tendo Achillis jerk rarely present.)

SPHINCTERS.—Unaffected.

ELECTRICAL REACTIONS.—May be simple diminution of excitability, stronger currents being necessary to produce contractions; or may be reaction of degeneration.

STEPPAGE GAIT.—When lower extremities affected. Due to foot-drop from weakness of extensors. *Foot lifted high* to clear toes from ground, thrown forward, and slapped flat on ground.

OTHER CHANGES may be:—

TROPHIC.—In late stages. Skin smooth and glossy. Nails brittle and cracked.

VASOMOTOR. Not common. May be œdema.

ATAXIA. Usually absent, but in rare cases (alcoholic) marked.

CONTRACTURES of muscles and resulting deformities may occur in chronic cases.

Neuritis may spread along nerve trunks, usually upwards; or distribution extend to fresh nerves, and muscles of respiration may be affected.

Alcoholic Peripheral Neuritis.—Commoner in women. Age, 30 to 40 years. Especially chronic tipplers. *Onset* gradual. Distribution symmetrical. (*See also* ALCOHOLISM.)

INITIAL SYMPTOMS.—Tingling in feet and hands. Twitching muscles. Cramps in calves.

GENERAL CHARACTERISTICS:—

1. Weakness and paralysis. Onset in lower extremities, mainly extensors and calf muscles, whence foot-drop and steppage gait. Later, hands and forearms.
2. Rapid wasting of affected muscles, mainly below knee.
3. Tenderness of muscles, especially of *calves*. Sometimes soles of feet.
4. Deep reflexes lost; knee-jerks and ankle-jerks absent.
5. Sensory changes variable. Lost in early stages. Numbness and tingling usual. Pains slight, or occasionally severe, and nerve-trunks tender. Some anæsthesia usual, partial or complete.

Alcoholic Peripheral Neuritis, *continued.*

6. Sphincters unaffected. (With mental changes incontinence may occur.)
7. Hands and feet become congested when hanging down.
8. Mental changes common, especially Korsakow's syndrome; also delirium, convulsions (*see* ALCOHOLISM).

Paralysis may extend, involving muscles of respiration. Face rarely affected.

PROGNOSIS.—Usually good, even in severe cases with long period before improvement commences. May be many months. Small muscles may remain wasted: steppage gait may persist. From mental symptoms, complete recovery is rare. Cardiac degeneration and pulmonary tuberculosis may be fatal.

DIAGNOSIS.—Especially from:—

1. ARSENICAL NEURITIS (*see below*).
2. TABES.—In rare forms of neuritis with marked ataxia. Note absence of pupil changes, sphincter troubles, and optic atrophy; presence of steppage gait and tender muscles, electrical reactions; congestion of hands.

Arsenical Multiple Neuritis (*see* ARSENIC POISONING).—Closely resembles alcoholic form. In diagnosis, note: (1) Other signs of arsenical poisoning, especially skin changes; (2) Commences in feet rather than in calves (thus differing from alcoholic); (3) Pain commoner and severer; (4) Progress and muscular atrophy more rapid.

Diphtheritic Multiple Neuritis (*see* DIPHTHERIA). Selective action on nerve-supply of eye muscles, palate, pharynx and larynx, and muscles of respiration.

Multiple Neuritis from Lead, Beri-beri, Leprosy, etc.—*See* LEAD, BERI-BERI, LEPROSY, etc.

Acute Febrile or Toxic Polyneuritis. A rapidly ascending polyneuritis. Some cases may be aberrant encephalitis lethargica.

ETIOLOGY.—Usually no obvious cause. Possibly cold, fatigue, etc.

ONSET.—Resembles acute specific fever: Sudden onset, rigors, pains in back and limbs, headache and malaise, high temperature.

SPECIAL CHARACTERS.—

1. PARALYSIS commences in legs, and rapidly ascends. May involve intercostals, and in fatal cases the diaphragm; face usually escapes; also sphincters (not invariably). Muscles flabby and waste very rapidly.
2. SENSORY CHANGES variable. Pain may be slight or severe, with or without anæsthesia.

PROGRESS.—Severe forms: death in 2 to 10 days. If patient survives initial period, prognosis is surprisingly good, but recovery needs 1 to 2 years.

DIAGNOSIS.—*See* LANDRY'S ACUTE ASCENDING PARALYSIS.

General Prognosis.—

WHEN CAUSE IS REMOVABLE, prognosis is good, even with extensive paralysis. Improvement may be slow at the com-

mencement, and occupy many months, with final recovery almost complete. Muscles recover in inverse order to involvement.

RESIDUAL CHANGES may be. (1) Permanent wasting and weakness of small muscles of hands, or of peronei; (2) Steppage gait; (3) Contractures; also (4) Mental changes in alcoholism.

IN ACUTE PROGRESSIVE FORMS, mortality varies with: (1) Rapidity of extension; (2) Involvement of respiratory muscles. Mortality high from latter.

General Diagnosis.—Usually simple, by the characteristics: (1) Symmetrical; (2) Flaccid paralysis; (3) Muscular wasting, (4) Reflexes absent; (5) Reaction of degeneration present; (6) Tender muscles; (7) Sensory changes; (8) Sphincters unaffected; (9) No pupil changes. Ataxia rare. Diagnosis from:—

IN EARLY STAGES. Acute rheumatism.

IN CHRONIC FORMS.—Progressive muscular atrophy. Multiple neuritis is characterized by more rapid onset, wide distribution, sensory changes, tenderness of muscles.

IN ACUTE FORMS.—(1) Acute myelitis of lumbar enlargement. (Sphincters affected; pyrexia. When myelitis above lumbar enlargement, symptoms are of upper motor neuron lesion, and anæsthesia extends to trunk.) (2) Acute poliomyelitis. (Paralysis less symmetrical; proximal segments may be affected.) (3) Landry's acute ascending paralysis (q.v.) (4) Tabes. (5) Trichinosis.

Treatment.—

GENERAL.—

REMOVE THE CAUSE.

REST IN BED.—Essential 3 to 6 weeks or more. Also saves heart and respiratory muscles.

DIET. Generous.

Water-bed preferable. Wrap limbs in cotton-wool.

SPECIAL INDICATIONS IN EARLY STAGES.—

PAIN.—Often eased by cradle and arrangement of limbs. Hot fomentations.

Drugs: Phenacetin, aspirin; morphia as last resort.

SLEEPLESSNESS.—Bromides or paraldehyde.

CARDIAC WEAKNESS (especially in alcoholics).—Digitalis and strychnine.

PREVENTION OF CONTRACTURES AND STRETCHING OF MUSCLES—e.g., foot-drop and contraction of hamstrings.—Very important. By splints, celluloid splints, and sand-bags.

IN LATER STAGES.—

MASSAGE AND PASSIVE MOVEMENTS.—Not in acute stages, but commence when calves are less tender, or wasting begins.

ELECTRICAL TREATMENT.—As in MASSAGE.

As MUSCLES RECOVER, *encourage* i. in playing draughts, etc.

DRUGS.—Tonics of iron and strychnine. Arsenic in all cases except arsenical neuritis. Sodium salicylate and potassium iodide may ease pain in early stages.

III DISEASES OF THE SPINAL NERVES.

1. CERVICAL PLEXUS.

Phrenic Nerve.—

CAUSES OF PARALYSIS —

(1) Fractures, tumours, etc, of spine involving anterior horns of 3rd and 4th cervical segments or the nerve roots

(2) Diphtheritic neuritis, rarely alcoholic or lead neuritis

(3) Wounds tumours in neck (Never in thoracic aneurysm)

Occasionally no obvious cause (unilateral only)

CHARACTERISTICS —*Diaphragm paralyzed* Results —

(1) Respiration by intercostals and accessory muscles Epigastrium drawn in during inspiration Often noticeable only on deep respiration Lower intercostals may also be paralyzed

(2) Dyspnœa on slight exertion. Respiration at rest usually normal

(3) Congestion at base of lungs usual rales and diminished resonance

(4) X rays show deficient movement of diaphragm

(5) Presence of Litten's sign, i e absence of the normal undulations in 6th to 10th spaces in axilla (due to movement of diaphragm)

PROGNOSIS in bilateral forms Bad respiratory failure or pneumonia

DIAGNOSIS Difficult especially in women In unilateral lesions affected side of the thorax moves more than the normal side Deficient movement occurs from inflammation above or below diaphragm

Hiccough.—Involves spasm of (1) Diaphragm, (2) Glottis

CAUSES —

(1) IRRITATION OF DIAPHRAGM e g pepper hot fluids gastric distention

(2) IDIOPATHIC

(3) REFLEX in abdominal diseases peritonitis (general or local), intestinal obstruction enteric dysentery etc.

(4) HYSTERIA Also in intracranial tumours and disease

TREATMENT —

MILD FORMS —Holding the breath long draught of water peppermint water and carminatives

SEVERE FORMS (last two groups) Often obstinate Blister to epigastrium firm traction on tongue for one to two minutes bromides, faradization of epigastrium or phrenic nerve Most effective are injections of morphia chloroform inhalation, or anæsthesia

2. BRACHIAL PLEXUS.

Lesions usually affect a portion only, rarely entire plexus Causes are almost all traumatic, either supraclavicular (usually laceration) or infraclavicular (mainly compression) Lesions of the plexus are considered first; then those of the separate branches

Lesions of the Plexus.—

CAUSES.—

1. TRAUMA TO NECK.—Blows, wounds, falls on neck, or violent drag on arm.
2. OBSTETRICAL PARALYSIS.—In this and the first group the injury may stretch or tear the plexus, either all, or frequently only the upper cord.
3. FRACTURES AND DISLOCATIONS compressing cords—e.g., dislocated humerus, fractured clavicle, or resulting callus.
4. CERVICAL RIB.—Especially affects lower cord.

Rare causes are: Tumours; subclavian aneurysm in neck, toxic neuritis.

VARIETIES.—(1) Complete. (2) Partial—common forms being: (*a*) Upper arm type, C5 and C6 (Erb-Duchenne paralysis); (*b*) Lower arm type, C8 and D1 (Klumpke's paralysis).

1. COMPLETE LESION. Arm hangs to side. Results in:—
   (i) Complete motor and sensory paralysis of extremity. Serratus magnus, rhomboids, and levator anguli scapulæ escape. Flaccid paralysis of lower motor neuron type.
   (ii) Pupil small and palpebral fissure narrowed (branch of D1 to sympathetic system). May be slight enophthalmos.
2. PARTIAL LESION.—
   a. UPPER ARM TYPE: ERB-DUCHENNE PARALYSIS.—(Obstetrical palsy.)
      Lesion: 5th cervical root, and sometimes also 6th; usually rupture.
      Muscles affected: Deltoid, biceps, brachialis anticus, supinator longus, may be others, varying with lesion.
      Position of arm: Hangs at side, rotated inwards.
      Movements lost: (i) Abduction of arm; (ii) Flexion of elbow; (iii) Supination of hand.
      Sensory changes: Slight.
   b. LOWER ARM TYPE: KLUMPKE'S PARALYSIS.—(Usually from compression, or residual from extensive paralysis of plexus.)
      Lesion: 8th cervical and 1st dorsal root.
      Muscles affected: (i) Intrinsic muscles of hand; may also affect (ii) Flexors of wrist and fingers.
      Motor changes: Claw hand develops. If flexors are affected, inability to grasp.
      Sensory changes: Numbness or anæsthesia, inner side of forearm and hand.
      Eye changes: Pupil contracted, palpebral fissure narrowed, slight enophthalmos, on side of lesion. May be absent.

Cervical Ribs.—

GENERAL CONSIDERATIONS.—

1. Ribs usually bilateral, with unilateral symptoms, commonly on left.
2. Symptoms in 5 to 10 per cent only.
3. Onset in early adult life; generally women.
4. Numerous varieties of ribs, from rudimentary to well-

Diseases of the Spinal Nerves - Cervical Ribs, *continued.*

formed. Similar symptoms occur, rarely, with an abnormal first dorsal rib.

Late onset is due to dropping of shoulder and not to late ossification of rib.

SYMPTOMS.—Due to compression of 1st dorsal nerve, 8th cervical nerve (to less extent), and artery :—

1. *Pain.*—Initial, commonest, and may be sole, symptom. Pain, numbness and tingling in ulnar border of forearm to wrist and fingers. Sensory changes often slight.
2. Motor symptoms.—Wasting and paresis of intrinsic muscles of hand.
3. Weak radial pulse, strengthening on lifting arm. Not common.

Occasional symptoms : Dissociated anæsthesia. Vasomotor changes. Pains in neck and back of head. Subclavian aneurysm. Thrombosis. Pupillary changes.

DIAGNOSIS.—By palpation and X rays. Always consider in wasting of one hand.

TREATMENT.—Massage. Electricity. Removal of rib : results good (but some authorities disagree).

Long Thoracic Nerve (nerve of Bell—from 5th, 6th, and 7th cervical roots).—Supplies the serratus magnus.

SERRATUS MAGNUS PARALYSIS.—Isolated paralysis from :—

(1) Injury in neck : especially carrying weights. Also rarely by wounds in axilla, or violent contraction of scalenus medius.

(2) Primary neuritis. Cold.

It also occurs, with other lesions, in dystrophies and progressive muscular atrophy.

FUNCTIONS OF MUSCLE.—(1) Draws scapula forward ; (2) Rotates inferior angle up and forwards.

SYMPTOMS.—

1. With arms at rest : deformity slight ; inferior angle of scapula slightly prominent and tilted towards spine.
2. When arms are held horizontally : scapula becomes 'winged'.
3. Impairment of power of pushing.
4. Arms cannot be raised above horizontal.

No sensory changes in pure lesions. Trapezius often affected.

Circumflex Nerve.—

CAUSES OF PARALYSIS.—(1) *Injuries to shoulder*, by dislocation, fracture, or blows ; by crutches ; occasionally during operations. (2) *Arthritis*, inflammation spreading from joint. (3) From cold. diabetes, lead, etc. : very rarely.

MUSCLES SUPPLIED.—Deltoid ; teres minor. A branch is sent to the shoulder-joint.

SYMPTOMS.—

1. Arm cannot be raised or rotated outwards.
2. Wasting over shoulder.
3. Pain, often severe, and impaired sensation over shoulder.

(4) Groove between head of humerus and acromion, from relaxation of joint.

In chronic cases, adhesions may form in joint.

DIAGNOSIS.—From joint disease. Note electrical reactions. Suprascapular nerve often affected also.

Musculospiral Nerve.—Paralysis common.

CAUSES.—(1) *In axilla*: Dislocations, fractures, callus, crutches. (2) *In course round humerus*: Pressure between bone and hard substance—e.g., sleeping with arm over back of a bench. Occasionally: Neuritis from cold. Rarely: Strong contractions of triceps. *Lead palsy* affects certain branches.

SYMPTOMS.—

CHARACTERISTIC SYMPTOM.—Radial paralysis: 'wrist-drop' and inability to extend fingers at metacarpo-phalangeal joints: from paralysis of extensors of wrist and fingers. (Interossei unaffected.)

OTHER SYMPTOMS.—When injured in axilla, triceps, brachialis anticus, and supinator longus also affected, with loss of extension at elbow and supination, but latter effected by biceps when elbow flexed.

SENSATION.—Numbness and tingling in distribution, mainly radial side of hand. Sensory changes variable, often absent, or anæsthesia of radial branch.

ELECTRICAL REACTIONS.—In pressure palsies may be normal below site of injury, but nerve inexcitable above.

DIAGNOSIS.—Usually simple: lesion unilateral. In lead palsy, lesion is bilateral and supinator longus escapes.

PROGNOSIS.—Pressure palsies usually recover in a few days; permanency is rare.

Ulnar Nerve.—

CAUSES OF PARALYSIS.—(1) *Wounds of forearm*. (2) *Injuries at elbow-joint*: symptoms may commence after long interval. Rarely: nerve dislocated from groove at olecranon; neuritis from cold, etc. Always affected in leprosy.

MUSCLES SUPPLIED.—Flexor carpi ulnaris and ulnar half of flexor profundus digitorum (branches in forearm); interossei; two inner lumbricals; small muscles of little finger; adductor of, and inner head of short flexor of, thumb.

RESULTS OF LESION.—Vary with site:—

1. AT OR ABOVE ELBOW.—(a) Flexion of wrist feeble and incomplete; attempt causes radial deviation. (b) Wrist hyperextends on straightening fingers. (c) Fingers extended at metacarpo-phalangeal joint and flexed at others: in index and middle fingers less marked, owing to escape of lumbricals (median nerve). (d) All movements of little finger lost. (e) Separation of fingers lost. (f) True adduction of thumb lost.

2. LESION NEAR WRIST.—'*Claw hand*' rapidly develops, from escape of flexor profundus digitorum and unopposed action of long flexor and extensors (index and middle fingers rather less than others). Thumb abducted.

Diseases of the Spinal Nerves—Ulnar Nerve, *continued.*

MUSCULAR WASTING marked: hypothenar eminence, interosseal spaces, and between thumb and index finger.

LOSS OF SENSATION.—Over 5th finger and ulnar half of 4th finger and pulp and nail. Protopathic sensation lost over smaller area, and deep sensibility present. When lesion is below dorsal cutaneous branch, loss is much less extensive (Sherren).

DIAGNOSIS.—From lesions of lower cord of brachial plexus. Characteristics are: (1) Claw-hand; (2) Muscular wasting; (3) Sensory changes.

Median Nerve.—Rarely affected alone.

CAUSES OF PARALYSIS.—(1) Wounds on palmar surface of wrist. (2) Wounds in forearm or arm. Occupation palsies affect muscles supplied by median nerve.

RESULTS OF LESION.—These vary with site.

1. LESION AT ELBOW.—Movements lost are. Pronation of forearm; flexion of wrist (feebly present, with ulnar deviation); flexion of all interphalangeal joints except two distal joints of two ulnar fingers; abduction of thumb.
2. LESION AT WRIST.—Thumb movements mainly affected, especially abduction.

MUSCULAR WASTING of thenar eminence.

LOSS OF SENSATION.—Variable. On palmar aspect, three and a half fingers; on dorsum, last two phalanges of index and middle fingers and radial half of 4th finger (Sherren). Protopathic loss of slightly less extent. Deep sensibility retained.

3. LUMBAR AND SACRAL PLEXUSES.

Lesions of nerves of lower limb are rarer than those of upper limb.

Obturator Nerve.—Isolated lesion rare.

CAUSES.—Injuries occur in parturition. Rarely from pelvic growths or obturator hernia.

RESULTS OF LESION.—

LOSS OF MOVEMENTS.—(1) Adduction of thigh (leg cannot be crossed over other); (2) Outward rotation (obturator externus).

LOSS OF SENSATION, OR PAIN.—Inner side of lower half of thigh. Often indefinite.

Anterior Crural Nerve.—Isolated lesion rare.

CAUSES.—(1) Dislocation and fractures of femur, or wounds in groin. (2) Psoas abscess, or abdominal growth. Rarely in parturition (usually with obturator nerve).

RESULTS OF LESION.—

LOSS OF MOVEMENTS.—Extension of knee. Walking is possible.

WASTING of quadriceps muscle, with reaction of degeneration.

KNEE-JERK absent.

LOSS OF SENSATION, OR PAIN.—(1) Lower two-thirds of anterior and inner side of thigh; (2) Inner side of leg to big toe (internal saphenous).

CONTRACTURE of flexors occurs, if neglected

Note.—Injury near plexus: psoas also affected, with loss of flexion of thigh. Somewhat lower: psoas escapes, but flexion weak, from paralysis of iliacus.

DIAGNOSIS - From wasting in hip joint disease · no reaction of degeneration.

Superior Gluteal Nerve.—Loss of abduction of thigh.

External Cutaneous Nerve.—'*Meralgia paræsthetica*' is characterized by pain and paræsthesia on front and outer side of thigh, may be severe, sensory changes slight

Commoner in males in females usually in pregnancy

Probably neuritis from trauma in course under Poupart's ligament near anterior superior spine of ilium

TREATMENT. Excision of nerve. Sometimes fails.

Sacral Plexus.—Lesions not common

CAUSES.—Lesions may arise from (1) Pelvic tumours, caries, or inflammation. (2) Parturition · fœtal head compresses higher ... against l im of pelvis (i e, mainly external popliteal fibres) (3) Rarely, neuritis extending from sciatic nerve

SYMPTOMS.—Resemble incomplete sciatic paralysis, but may include (1) Gluteal and external rotators of thigh, if upper roots involved, (2) Anæsthesia on the back of thigh, buttocks, and perineum, if lower roots involved (small sciatic nerve).

Sciatic Nerve.—

CAUSES OF PARALYSIS.—(1) Fractures of pelvis or femur, or dislocations (2) Wounds in leg (3) Parturition. (*a*) In mother, (*b*) In infant from traction on leg Nerve divides into two main branches high in thigh, and one may escape in wounds

RESULTS OF LESION.—

LOSS OF MOVEMENTS.—(1) Injury at notch paralysis of (*a*) flexors of knee, (*b*) muscles below knee. (2) Injury below middle of thigh. flexors of knee escape

LOSS OF SENSATION Outer half of leg, and all the foot, except small area on inner side of dorsum.

WASTING OF MUSCLES

TROPHIC CHANGES not uncommon.

External Popliteal Nerve.—

CAUSES OF PARALYSIS.—(1) Trauma in course round fibula. (2) Wounds in popliteal space Occasionally: (3) Prolonged kneeling—nipped by biceps cruris tendon. Rarely: (4) Neuritis: primary or lead poisoning, tibialis anticus usually escaping

MUSCLES SUPPLIED.—Peronei, long and short extensors of toes; tibialis anticus

RESULTS OF LESION.—

MOTOR CHANGES.—(1) *Foot-drop*; (2) Toes flexed, (3) Foot commonly inverted, especially if tibialis anticus escapes, but varies with muscles affected. Sequels: (*a*) Steppage gait; (*b*) Talipes equinus (if internal popliteal unaffected).

LOSS OF SENSATION.—Outer half of front of leg and dorsum of foot to end of proximal phalanges of toes.

Diseases of the Spinal Nerves, *continued*

Internal Popliteal Nerve.—Course protected and injury rare.

RESULTS OF LESION—

LOSS OF MOVEMENTS—(1) Extension of foot, (2) Flexion of toes, whence inability to stand on tip toe. Foot is everted (by peroneus longus). Sequelæ—(*a*) Talipes calcaneo-valgus, (*b*) Claw foot, from contractures.

LOSS OF SENSATION.—Outer side and back of lower third of leg. Sole, and entire distal phalanges of toes.

Diagnosis of Lesions of Sciatic Nerve and Branches.—From: (1) Cauda equina, and sacral segments of cords. In these (*a*) Lesions are bilateral; (*b*) Sphincters are affected, (*c*) When lesion is in cord all roots are affected below level of disease. (2) Sacral plexus. Distribution of paralysis and anæsthesia.

IV. NEUROMATA.

A tumour connected with nerves may be—

1. TRUE NEUROMA.—Formed of nerve tissue. (*a*) Nerve fibres, (*b*) Ganglion cells. Latter is very rare; strictly the only true neuroma.
2. FALSE NEUROMA.—Formed of fibrous tissue. Intermediate forms are frequent. Malignant neuroma is very rare.

Plexiform Neuromata.—Multiple tumours on the nerves, due to hypertrophy of connective tissue. Often congenital and hereditary. No pain *per se*, but may compress other structures. Trigeminal nerve most often affected.

Von Recklinghausen's Disease (*Molluscum Fibrosum, Generalized Neurofibromatosis*).—

CHARACTERISTICS—

(1) Soft, fibrous nodules in skin, sessile or pedunculated, varying in number and size. May become enormous. Scattered over trunk and scalp, rare on hands or feet. Are neuro fibromata of cutaneous nerves.

(2) Plexiform neuromata on nerve trunks. May occur within spine and cranium.

(3) Patches of pigmentation. Pigmented nævi in some cases.

(4) Sensory and motor symptoms various: pain, paralyses, etc. Mental symptoms common: depression, and loss of intellectual power.

ORIGIN.—Probably from sheath of Schwann (this is absent in optic and olfactory nerves, where nodules never occur).

PROGNOSIS.—Depends on removal of tumours producing symptoms.

Note.—Several conditions are probably included in this group. May be congenital, and occasionally hereditary.

Tuberculosis Dolorosa.—Painful subcutaneous tubercles. Very small; directly below skin, solitary or multiple, commonest on face, chest, and near joints. Extremely tender. Are on terminal cutaneous nerves. Usually neurofibromata.

TREATMENT.—Excision.

Amputation Neuromata.—On central end of nerves divided by injury or operation. Very painful.
TREATMENT.—Removal; but may recur.

V. SCIATICA.

Pain in distribution of sciatic nerve arises from: (1) Neuritis; (2) Pressure on nerve or roots by tumours, etc.; (3) Neuralgia or neurosis.

1. NEURITIS.—Cold or wet: common causes. Gout. Rheumatism, especially spondylitis. Common in alcoholics. Occasionally in diabetes (bilateral); gonorrhœa, syphilis. Trauma rare.
2. ORGANIC CAUSES—Pelvic tumours—e g, uterine, fœtal head, or even full rectum.

Etiology.—Common in males. Rare under middle age.

Morbid Anatomy.—Interstitial neuritis. Nerve red and swollen.

Symptoms of Neuritis.—

1. PAIN IN COURSE OF NERVE.—Characters: (a) Unilateral; (b) Onset gradual, usually in upper thigh; at first following exertion or in positions stretching nerve. (c) Becomes constant, often with paroxysms, worse at night, on walking, or any sudden movement. (d) Severity increases and distribution extends; may involve entire nerve.
2. NERVE TENDER ON PRESSURE.—Especially over sciatic notch, mid thigh, popliteal space, and outer side of fibula.
3. PAIN ON STRETCHING NERVE (Lasègue's sign).—Flex hip and extend knee.
4. MUSCULAR WASTING, in chronic cases. Moderate degree. No reaction of degeneration.
5. NO CUTANEOUS HYPERÆSTHESIA, except over nerve trunk.

OTHER FEATURES.—*Walks* with knee bent, on toes, to relax nerve. *Knee-jerks* usually brisk. *Skin* generally dry and cold, occasionally sweating and trophic changes. *Cramps* and *spasms* not very common: usually at night. *Scoliosis* may develop: concavity usually away from affected side. Herpes rare.

Course.—Duration variable, often obstinate. Remissions common.

Diagnosis.—Pelvic tumours, lesions of spine, hip-joint, and spinal cord, etc., must be excluded. Rectal examination is important.

1. PELVIC TUMOURS, PELVIC DISEASES, ETC.—Note: (a) Area of sensory changes; (b) Reaction of degeneration and advanced muscular atrophy; (c) Absence of knee-jerks. Nerve trunk is not tender. Note *sacro-iliac disease*.
2. HIP-JOINT DISEASE.—Pain on *rotating thigh* or pressure on trochanter. Nerve trunk not tender.
3. LUMBAGO.
4. TABES.
5. INTERMITTENT CLAUDICATION.—After exertion only. Distribution not of nerves.
6. LESIONS OF CAUDA EQUINA.—Bilateral. Sphincters affected. Sensory changes.

Sciatica—Diagnosis, *continued*

Bilateral sciatica suggests general and not local cause (diabetes, gonorrhœa, syphilis, etc).

Treatment.—

APPROPRIATE TREATMENT OF SPECIAL CAUSES.—Gout, gonorrhœa, syphilis, etc

BOWELS OPENED REGULARLY.

REST IN BED WITH LONG BACK SPLINT —Advisable in all cases, for a few days to 3 to 6 weeks

HEAT ALONG COURSE OF NERVE —Hot bottles, hot sand bags, hot iron, or baths

COUNTER-IRRITATION —Blisters, cautery along nerve

DRUGS -- Often ineffectual Best are alkalis, sodium salicylate, and potassium iodide In acute pain, phenacetin, aspirin, etc, morphia as last resort.

ELECTRICITY. — Effects variable. Galvanic current Also kataphoresis

HYDROTHERAPY, SPA TREATMENT —Advantageous in elderly patients

INJECTIONS INTO NERVE Insert needle into nerve near sciatic notch, about 2 inches deep causes pain in distribution Inject a few c c. of 2 per cent novocain, then, slowly, 50 to 100 c c. of warm normal saline Repeat weekly, two or three times (Distilled water also used)

ACUPUNCTURE —Insert 6 needles into nerve at intervals leave 15 to 20 minutes

NERVE-STRETCHING

MASSAGE.—*In chronic cases,* to strengthen muscles

CHAPTER CXXV.

DIFFUSE AND LOCAL DISEASES OF THE SPINAL CORD.

I. MYELITIS.

By analogy with the meaning usually involved in its termination, *myelitis* should denote inflammation In general, it is applied in a wider, and etymologically more correct, sense to disease of the spinal cord.

Etiology.—

1. IDIOPATHIC — Cold, exposure, etc, possible factors
2. SYPHILIS.—Only cause of a *chronic* myelitis, others are acute or subacute. (This form is not referred to in this section *See* SYPHILIS OF THE NERVOUS SYSTEM)
3. COMPRESSION MYELITIS.—Commonly from caries, trauma, tumours, aneurysm. (*See* p. 778)
4. TRAUMA.—Fracture of spine. Rarely, concussion without fracture.
5. ACUTE SPECIFIC FEVERS —*Rare*: most often in enteric and

influenza. *Very rarely*, in gonorrhœa, measles, dysentery, and other specific infections.

6. TUMOURS OF CORD, MENINGITIS OF CORD.—*Rare.*
7. ACUTE POLIOMYELITIS.

Morbid Anatomy.—Lesion may be: (1) *Transverse myelitis*: of vascular origin; thrombi marked. (2) *Diffuse* or *disseminated myelitis*, extending over wide or scattered areas: of inflammatory origin; leucocytic infiltration marked.

MACROSCOPIC CHANGES.—Cord swollen and soft: in severe forms, diffluent. Meninges injected. On section, no distinction between white and gray matter; often hyperæmia.

HISTOLOGY.—(1) Nerve cells: swollen and irregular, nuclei degenerated, cytoplasm granular and fatty. (2) Nerve-fibres: myelin sheaths swollen, do not take Weigert-Pal's stain, and show fatty degeneration (Marchi's method); axis cylinders irregular and degenerate later. (3) Blood-vessels: distended; thrombi common. Perivascular lymph spaces infiltrated with cells, mainly mononuclear.

LATER STAGES.—Usually less acute forms. Two characteristics: (1) *Sclerosis* of affected area, by proliferation of neuroglia; (2) *Ascending and descending degenerations*, tracts later becoming sclerosed.

Clinical Types of Acute Myelitis.—(1) Acute transverse myelitis: (a) Dorsal; (b) Cervical; (c) Lumbo-sacral. (2) Acute diffuse or ascending myelitis, disseminated myelitis.

1. **Acute Transverse Myelitis.**—

*a. **Dorsal Myelitis.**—Commonest form; usually about 9th to 8th dorsal segment.

ONSET rapid; symptoms at maximum within few days.

INITIAL SYMPTOMS may be either: (1) Motor: weakness and stiffness in legs. (2) Sensory: numbness, tingling, or aching. (3) Sphincters: difficulty in micturition. Constitutional symptoms (pyrexia, etc.) slight or absent.

SYMPTOMS.—

1. PARALYSIS OF LOWER LIMBS.—Complete or partial; in latter case, flexors mainly affected. Of upper motor neuron type (spastic paraplegia)—viz., wasting only from disuse, and electrical reactions normal or diminished. Lower trunk may be affected, in which case umbilicus moves upwards on contracting abdominal muscles.
2. SENSORY CHANGES.—(a) *Anæsthesia*: usually to level of lesion, but light touch lost over smaller area than pain: upper limit definite or indefinite. (b) *Hyperæsthesia*: band common at upper level. Also girdle pains.
3. DEEP REFLEXES.—*Early* stages: diminished (flaccid). *Later*: spastic, knee-jerks increased, ankle-clonus and Babinski's sign present. *Superficial reflexes*: lost to level of lesion.
4. SPHINCTERS usually affected. Either: (a) Retention with overflow; or (b) Bladder empties periodically. May be unconsciousness of passage.

Dorsal Myelitis—Symptoms, *continued.*

5. TROPHIC CHANGES liable to occur: œdema, bullæ, *bedsores*. *Cystitis* may develop from retention, catheterization, and infection of urine.

With a complete transverse lesion or a grave injury, all reflexes are lost, 'stage of flaccidity'. This may be permanent; but, in absence of sepsis, such stage of shock may be succeeded, in about three weeks, by a 'stage of reflex activity' a slight stimulus will now produce violent spasms of limbs, trunk, bladder, etc., the so-called 'mass reflex' (Head and Riddoch).

PROGNOSIS.—Improvement variable in each group of symptoms. Complete recovery rare. Patient usually arrives at a stationary stage in condition of lateral sclerosis, with marked muscular rigidity, tendency to spasms, increased reflexes, but slight wasting or electrical changes. Recovery of sphincter control variable. *Contractures* of flexor muscles develop

b. **Cervical Myelitis.**—*Rare*, except in trauma. Diaphragm often involved (phrenic nerve), with rapidly fatal result.

SYMPTOMS.—

1. LOWER LIMBS, REFLEXES, ETC.—Condition as in dorsal myelitis, *upper motor neuron paralysis.*
2. UPPER LIMBS.—*Lower motor neuron paralysis* i.e., with *muscular wasting*, etc.
3. ANÆSTHESIA.—Lower limbs and trunk; in upper limbs depends on segment involved.
4. SPECIAL SYMPTOMS may be present: pupil small (spinal myosis), slow pulse, vomiting, hiccough; occasionally hyperpyrexia.

c. **Lumbar and Lumbo-sacral Myelitis.**—Uncommon.

SYMPTOMS.—

1. WEAKNESS AND PARALYSIS OF LEGS. Type, extent, and distribution vary with site of lesion: partly of upper and partly of lower motor neurons, but mainly of latter from destruction of anterior horn cells—whence: (*a*) *Atrophy* of muscles; (*b*) *Knee-jerks absent*, with ankle-clonus and Babinski's sign present.
2. SENSORY CHANGES.—Not above groin
3. SPHINCTERS affected: urine dribbles (true incontinence)
4. TROPHIC CHANGES, *bedsores* and *cystitis*, are early and severe.

2. **Acute Ascending or Diffuse Myelitis.**—*Rare* Onset and progress rapid; usually commences in legs.

SYMPTOMS.—

1. PARALYSIS AND ANÆSTHESIA, progressively ascending.
2. PYREXIA and constitutional symptoms from onset.
3. SPHINCTERS paralysed.
4. TROPHIC CHANGES.—Rapid *wasting*, *bedsores*, and *cystitis*.

DEATH.—Usually within 5 to 10 days.

ORIGIN.—Probably infective.

DIAGNOSIS.—Usually simple. From other acute progressive paralyses:—

1. LANDRY'S PARALYSIS: By *sensory*, *sphincter*, and trophic changes, wasting, and *pyrexia*.
2. ACUTE MULTIPLE NEURITIS: By *sphincter* affection, completeness of anæsthesia, and absence of muscular pain.

Rare Types.—

DISSEMINATED MYELITIS.—Brain and cranial nerves also affected. Scattered lesions and irregular symptoms.

DIFFUSE CENTRAL MYELITIS.—Rapid paralysis, anæsthesia, and trophic changes Reflexes absent. Commences in arms or legs. Fatal.

Prognosis.—

GENERAL PRINCIPLES:—

1. No definite prognosis of amount of recovery can be given at onset. Improvement often rapid to a stage and then stationary. Complete recovery very rare.
2. Better when following some *definite* illness.
3. The greater the extent of the symptoms, the worse the prognosis
4. *Anæsthesia*. a *sharp* upper margin and extension to level of lesion is worse than indefiniteness.
5. Best in *dorsal* myelitis. In *cervical* lesion, frequent fatal respiratory paralysis or disease. In *lumbar* lesion, sphincters and legs rarely recover: mortality high from bedsores and cystitis.
6. *Transverse type* better than diffuse and ascending.
7. High mortality in *bedsores* and *cystitis* (result in sepsis and pyelonephritis).

Diagnosis of Transverse Myelitis.—Usually easy. Characterized by: (*a*) *Paralysis and anæsthesia* up to a fairly definite level; (*b*) *Sphincters* affected; (*c*) *Increased reflexes* (exceptions noted above). Diagnosis from:—

1. CONDITIONS WHERE THE SYMPTOMS ARE DUE TO AN EXTRASPINAL CAUSE.—
   a. CEREBRAL LESIONS. Excluded by bilateral nature and anæsthesia.
   b. ACUTE MULTIPLE NEURITIS.—Difficulty only in lumbosacral lesions. In neuritis: (i) Pain greater and muscles tender, (ii) Paralysis flaccid; (iii) *Anæsthesia* slighter, and corresponds to peripheral nerves and not segments; (iv) *Sphincters* unaffected (unless mental disturbance).
   c. HYSTERICAL PARAPLEGIA.—Other signs of hysteria; never an extensor plantar response; bilateral anæsthesia very rare. (May coexist with myelitis)

   Rarely:
   d. DISSEMINATED SCLEROSIS. Acute onset rare, and no anæsthesia.
   e. LANDRY'S PARALYSIS.—*See* ASCENDING MYELITIS.
2. OTHER INTRASPINAL LESION.—
   a. SYPHILIS (q.v.).—History, rash, etc. Wassermann reaction (blood and serum), and cerebrospinal fluid.
   b. COMPRESSION OF CORD.—Local tenderness and deformity in

Diagnosis of Transverse Myelitis, *continued.*

back; slow onset; root symptoms often severe; X rays. *In tumours,* primary growth—e g, breast.

c. HÆMORRHAGE INTO CORD.—Very rare. Abrupt onset.

d. INTRAMEDULLARY TUMOURS.—Onset slower and unilateral. Dissociated anæsthesia and Brown-Séquard's paralysis.

e. SUBACUTE COMBINED DEGENERATION.—Anæmia. Reflexes absent. Slow onset.

Treatment.—*Special Indications*: (1) Prevent bedsores and cystitis; (2) Aid recovery of muscles and reduce contractures. Skilful nursing is essential.

ACUTE STAGE.—

1. REST IN BED, *on water-bed.* Frequent change of posture. Avoid burns from hot-water bottles.
2. SKIN kept absolutely dry and clean. If an area reddens, wash with spirit, dry, and dust with powder (zinc oxide and starch). Foment bedsores and treat as ulcers.
3. BLADDER.—(*a*) Retention: frequent catheterization, strictly aseptic; or 'expression of bladder'. (*b*) Incontinence: urinal. *Parts must be kept perfectly clean,* and wool packed round frequently. For cystitis, bladder washes and urotropine.
4. BOWELS regulated. Enema daily if necessary.

No local treatment to spine, or drug, is of proved value.

AFTER ACUTE STAGE (10 to 14 days).—

NOURISHING DIET. GENERAL TONICS: avoid strychnine if spastic.

ENCOURAGE TO MOVE LIMBS.—Massage and movements.

REFLEX SPASMS.—Bathe with hot water; sedatives, phenacetin, bromides, etc.

CONTRACTURES.—Watch for and counteract by arranging position of limbs.

II. COMPRESSION OF THE SPINAL CORD.

(*Compression Myelitis.*)

Compression myelitis is a term applied to symptoms and lesions resulting from slow compression of the spinal cord.

Causes.—(1) *Tuberculous caries*: commonest cause (2) *Fracture-dislocation* of spine. (3) *Tumours* of: (*a*) Vertebræ; (*b*) Meninges and roots; (*c*) Cord. (4) *Aneurysms* of aorta. Very rarely: (5) Arthritis deformans; (6) Syphilitic caries; (7) Pachymeningitis (*P. cervicalis hypertrophica*). Occasionally: Hydatid cysts and cysticercus.

Symptoms.—Result from affection of: (1) Vertebræ; (2) Nerve roots; (3) Spinal cord.

1. VERTEBRÆ.—(*a*) *Local pain and tenderness.* (*b*) *Rigidity* of back. (*c*) *Deformity.* The pain is increased by jarring and movement: may be present before deformity.
2. NERVE-ROOT SYMPTOMS.—At level of the lesion. From compression or irritation in canal or foramina. *Pain and*

hyperæsthesia in segments affected; often agonizing. Later there may be *anæsthesia* or *anæsthesia dolorosa* (i.e., pain felt over an anæsthetic area). Occasionally, atrophy of muscles from anterior-root affections. Rarely, herpes zoster.

3. CORD SYMPTOMS (*see* TRANSVERSE MYELITIS).—*Onset* usually very slow; rarely rapid, from: (*a*) Vascular disturbances—e.g., œdema; (*b*) Inflammation—i.e., true 'myelitis'.

Symptoms commence in lower limbs: a *spastic paraplegia*, as in transverse myelitis, characterized by: (*a*) *Weakness of legs* (earliest sign); (*b*) Rigidity; (*c*) *Increased knee jerks* and deep reflexes; Babinski's sign. Also, but *less marked* than in myelitis: (*d*) Sphincters affected; (*e*) Sensory changes and anæsthesia. Final symptoms vary with level of lesion (*see* TRANSVERSE MYELITIS). Bedsores not common.

ABSCESS FORMATION, psoas and retropharyngeal, in tuberculous caries.

Tuberculous Caries of Spine *causing compression.*—

ETIOLOGY.—

AGE.—Usually childhood, may be later.

PREDISPOSING FACTORS.—Tuberculosis elsewhere, e.g., lungs. Occasionally history of injury. Tuberculous family history common.

MORBID ANATOMY.—Commences in bodies of one or more vertebræ; softening, caseation, and collapse follow, with resulting deformity. Tuberculous mass forms in vertebral canal, but rarely penetrates dura, and very rarely directly invades cord.

CAUSE OF COMPRESSION.—(1) *Tuberculous mass* in canal usual cause. Rarely: (2) Compression by bony deformity; (3) Abscess; (4) Myelitis from circulatory or inflammatory changes (rapid progress).

Actual destruction of nerve tissue uncommon (hence recovery on treatment).

The nerve roots are affected in the canal or intervertebral foramina.

SYMPTOMS.—

SPECIAL CHARACTERISTICS:—

1. *Vertebræ.*—*Deformity* usually sharp, with a prominent spine: generally long preceded by *local pain, tenderness, and rigidity.* Compression of cord by tuberculous mass *in absence of deformity* may occur, but rare.
2. *Cord Symptoms.*—Onset usually late. Increased knee-jerks earliest sign, followed by weakness of legs.
3. *Root Symptoms.*—Rarely severe, and often absent.

CERVICAL REGION.—Frequent in axis and atlas. Common symptoms: (1) Spasm of cervical muscles; (2) Sympathetic nerve affected, pupil dilated, etc. (3) Retropharyngeal abscess. Cord symptoms frequently absent. Recovery, with rigidity of neck and much callus.

THORACIC REGION.—Commonest site. Deformity and, later, cord symptoms common. Psoas abscess.

LUMBAR REGION.—Resembles above, but knee-jerks may be absent.

Tuberculous Caries of Spine, *continued.*

COURSE AND PROGNOSIS.—General principles:—

1. Children better than adults.
2. Severe myelitis may recover under treatment.
3. Dorsal lesions better than cervical or lumbar.
4. Bad with tuberculous lesions elsewhere.
5. Compression by bone serious.

TREATMENT.—Absolute rest, with hyperextension and various mechanical appliances. General treatment for tuberculosis: fresh air, fats, etc. *Laminectomy*, especially in adults, if no improvement after long rest. Treatment for 12 to 24 months.

Tumours of the Vertebræ.—

PATHOLOGY.—

1. BENIGN.—Very rare. Exostoses, chondroma, etc.
2. MALIGNANT.—(*a*) Carcinoma: Always secondary; primary growth commonest in *breast*, occasionally uterus, stomach, etc. (*b*) Sarcoma: Rare, primary or secondary. Extension to meninges or cord very rare. Compression of cord often absent, but roots affected in intervertebral foramina.

SYMPTOMS.

SPECIAL CHARACTERISTICS:—

1. *Root Symptoms.—Early and marked*: progresses to agonizing paroxysms, often on the slightest movement. Site varies with lesion. Later, anæsthesia or anæsthesia dolorosa. Anterior roots less commonly affected spasm or, later, atrophy of muscles
2. *Vertebræ.*—Pain and tenderness usually severe. Deformity less angular than in caries (growth replacing bone). Growth may invade spinal muscles.
3. *Cord Symptoms —Often absent.* Onset may be slow or acute (vascular or inflammatory myelitis).

TREATMENT.—Laminectomy to relieve pressure on nerve roots. Morphia usually necessary. Course progressive.

Tumours of Spinal Cord and Membranes.—*See* p. 781.

General Diagnosis.—A radiograph should never be omitted.

1. TUBERCULOUS CARIES.—Characteristics: (*a*) Local pain and tenderness over spine; (*b*) Rigidity; (*c*) Deformity, often prominent spine; (*d*) Increased knee-jerks. Also: (*e*) Root symptoms rarely severe; (*f*) Abscesses. Diagnosis is most difficult in absence of deformity.

DIAGNOSIS FROM:—

Tumours of vertebræ: Deformity less sharp; root symptoms early and severe; radiograph. Primary growth (breast); absence of tuberculosis. Age.

Aneurysm: Age; Wassermann reaction; physical signs.

Tumours of spinal meninges: Root symptoms earliest; symptoms unilateral at onset; distribution and progress; no deformity.

Spondylitis deformans: Age. Radiograph. Widespread rigidity.

Pachymeningitis: Usually cervical; root symptoms severe and of long duration before cord affected; bilateral; progress very slow.

2. TUMOURS OF VERTEBRÆ.—Characteristics: (*a*) Early root symptoms, increased by movement; (*b*) Vertebral pain and tenderness; (*c*) Deformity curved or absent; together with: (*d*) Primary growth, commonly in breast; (*e*) Rapid emaciation.

DIAGNOSIS FROM: Caries; aneurysm; tumours of meninges; and pachymeningitis. Also from biliary and other colics; neuralgia and neuritis, e.g., intercostal.

3. TUMOURS OF MENINGES.—Characteristics: (*a*) Symptoms commence unilaterally; (*b*) Root symptoms early; (*c*) Cord symptoms also unilateral—paralysis and root symptoms on side of lesion, with main sensory changes on opposite side (may be typical Brown-Séquard's paralysis); (*d*) Later, symptoms bilateral; (*e*) No deformity.

CONDITIONS NOT INVOLVING COMPRESSION OR DEFORMITY.—

DIAGNOSIS FROM:—

Hysteria and neurasthenia: Tenderness of spine not localized; no affection of sphincters; no Babinski's sign. In hysteria: distribution of anæsthesia 'stocking' or 'glove'; other hysterical signs.

Amyotrophic lateral sclerosis: No sensory changes

Neuritis and neuralgias—e.g., intercostal, *sciatica, lumbago*: Very difficult. Often by progress. X rays. Tenderness over nerve trunks.

III. TUMOURS OF THE SPINAL MEMBRANES AND CORD.

Varieties.—(1) *Extramedullary* or *meningeal* tumours; (2) *Intramedullary* tumours. All rare, especially intramedullary.

1. EXTRAMEDULLARY TUMOURS.—Usually on dorsal or lateral surface. Two groups:—

a. EXTRADURAL.—

Origin.—Dura mater, vertebral periosteum, or intervening tissues.

Pathology.—Most frequent: (i) Sarcoma; (ii) Hydatid cysts. Rarely: Lipoma, fibroma, etc. Sarcoma alone invades cord. Carcinoma very rare, always secondary.

b. INTRADURAL.—Commoner.

Origin.—Dura mater, meninges, spinal roots.

Pathology.—Most frequent: (i) Sarcoma; (ii) Fibrosarcoma or fibroma. Rarely: Gumma, psammoma, lipoma, neuroma, etc. Sarcoma may be local, or a diffuse sarcomatosis surrounding cord: either primary or secondary.

Tumours of the Spinal Membranes and Cord, *continued.*

2. INTRAMEDULLARY TUMOURS.—Usually in cervical or lumbar regions.

Origin.—In cord (glioma), or more commonly invading from pia mater.

Pathology.—(i) *Tubercle*—most common; (ii) *Glioma*, or gliosarcoma; (iii) Sarcoma; (iv) Gumma, very rarely. Degenerations common. Glioma usually commences in gray matter.

TUMOURS OF VERTEBRÆ.—*See* COMPRESSION OF THE SPINAL CORD.

Note.—Symptoms vary greatly with the position and extent of the tumour.

Extramedullary Tumours.—

SYMPTOMS.—*Unilateral*, usually, in early stages; later bilateral.

EARLY SYMPTOMS:—

1. *Pain in Back.*—Probably meningeal.
2. *Root Symptoms.*—Pain, hyperæsthesia in affected segments: subsequently often lost when roots destroyed.

LATER:—

3. *Cord Symptoms.*—Onset slowly from compression, or rarely rapidly from myelitis. Characteristics: (*a*) Sensory changes; (*b*) Spastic paralysis below segment; (*c*) Atrophic paralysis in affected segment. *Unilateral* position of growth results in: (i) On side of tumour: paralysis and root symptoms. (ii) On opposite side: anæsthesia: of variable extent, but involves all sensations—i.e., is not 'dissociated'. Sphincters affected; reflexes increased; Babinski's sign present. Later, bedsores and cystitis common. Brown-Séquard's syndrome may be present typically, more often atypically (lesion not strictly unilateral).

Symptoms vary with position of tumour (*see* TRANSVERSE MYELITIS).

LOCALIZATION.—By area of root symptoms. Usually site is one segment above highest level of anæsthesia, but more accurately localized by minute sensory changes (Judson Bury).

NATURE OF GROWTH.—Rarely ascertainable. Note rate of progress, primary tumours, Wassermann reaction.

PROGNOSIS.—*Malignant*: rapidly fatal. *Benign*: many successful operations.

TREATMENT.—*Laminectomy* on diagnosis: to remove growth, relieve pressure, or divide roots. If inoperable (e.g., other growths present), morphia. Palliative treatment as in myelitis. With positive Wassermann reaction, syphilitic treatment, but early laminectomy if no rapid improvement (three to four weeks).

DIAGNOSIS.—From caries, tumours of vertebræ, pachymeningitis, and conditions not involving compression (*see* COMPRESSION MYELITIS). From intramedullary tumours: in latter, root symptoms absent, dissociated anæsthesia.

Intramedullary Tumours.—

SYMPTOMS.—*Unilateral* until late.

1. *'Dissociated' Anæsthesia.*—Pain and temperature sense lost on side opposite to lesion; light touch retained.
2. *Spastic Paralysis* on side of tumour. Sphincters affected; deep reflexes increased, and Babinski's sign present. Brown-Séquard's paralysis present typically or atypically. In segment of tumour, atrophic paralysis.
3. *Root Symptoms and Pain.*—Absent or slight.

PROGRESS.—Slow. Symptoms later bilateral, as in transverse myelitis.

TREATMENT.—Palliative, unless syphilitic.

Summary of Symptoms.—

1. EXTRAMEDULLARY.—(*a*) Unilateral onset; (*b*) Root symptoms early and severe; (*c*) Anæsthesia of all sensations on opposite side; (*d*) Paralysis on side of lesion; sphincters and reflexes affected.
2. INTRAMEDULLARY (*a*) Unilateral onset; (*b*) Root symptoms absent or slight; (*c*) Dissociated anæsthesia on opposite side; (*d*) Paralysis on side of lesion; sphincters and reflexes affected. Typical Brown-Séquard paralysis may occur.

IV. SYRINGOMYELIA.

(*Gliosis or Gliomatosis of the Spinal Cord.*)

A chronic disease of the spinal cord, characterized pathologically by new growth of neuroglia (gliosis) near the central canal, and presence of a cavity, and clinically by dissociated anæsthesia, trophic changes, and muscular atrophy.

Etiology.—

AGE AT ONSET.—Usually 20 to 30 years.

SEX.—Commoner in males, 2 to 1 female.

NO HEREDITARY AND NO SYPHILITIC FACTOR.

TRAUMA.—Previous severe injury to head, spine, ribs not infrequent, but influence as factor still uncertain; possibly causes hæmorrhage.

CONGENITAL ABNORMALITIES occasionally present.

Morbid Anatomy.—

SPINAL CORD.—On section, two characteristic changes are found:—

1. CAVITY present. Usually posterior to central canal. *Size*, variable, from slit to most of transverse section. Often extends into anterior, and less often into posterior, horns. May communicate with central canal, and is then lined with ependymal cells. Occasionally two cavities, but if so they are connected at some level.
2. GLIOSIS, increase of neuroglia around cavity. Of translucent gelatinous appearance, often blood-tinged. Consists of embryonic neuroglial tissue. Degeneration and hæmorrhage not infrequent.

Syringomyelia—Morbid Anatomy, *continued.*

The extent of these two changes, absolutely, and also relatively to each other, varies greatly. The *cavity* may extend up and down most of the cord. The *gliosis* may cover most of the section at certain levels, but the lateral white matter at the periphery is rarely affected. Vertically considered, the gliosis may extend beyond (above or below) the cavity, here forming a solid mass. In other cases the gliosis is limited to a small area surrounding the cavity. The lesions may extend into the bulb and 4th ventricle. Ascending and descending degeneration of affected tracts may occur.

SITE.—Commonest in lower cervical region; next in lumbar segments.

Pathogenesis.—Theories of origin include: —

1. A 'gliosis' or 'gliomatosis'—i.e., proliferation of neuroglia, with subsequent degeneration forming a cavity. Supported by invariable presence of some degree of gliosis.
2. A congenital defect, the cavity being remnant of an embryonic fissure. Supported by the occasional presence of other congenital defects, and by the embryonic nature of neuroglia.

Possibly both groups occur. Hæmatomyelia is improbable as a frequent origin.

Symptoms.—Result from destruction of nerve tissue and also from pressure on tracts, and hence vary greatly in extent and in combination of the three groups described.

ONSET.—Insidious. *Initial symptoms* noticed may be tingling and pains: frequently absence of pain following burns, cuts, etc.

THREE CHARACTERISTIC GROUPS OF SYMPTOMS—(1) '*Dissociated anæsthesia*'; (2) *Trophic changes* in skin, joints, and other tissues; (3) *Muscular atrophy* and motor changes. Upper extremities and trunk most commonly affected.

1. SENSORY CHANGES.—'*Dissociated anæsthesia*', viz., loss of sensations of heat, cold, and pain, with retention of light touch. Limits sharply defined; distribution usually asymmetrical and irregular, corresponding to segments or parts of segments. Heat, cold, and pain equally affected; or, slightly, heat more than cold, and cold than pain. Due to injury to fibres while *crossing cord in posterior commissure* (*see* p. 756).
2. VASOMOTOR AND TROPHIC CHANGES.
   Skin.—Thin and glossy, often sweating; hair on area diminished; nails furrowed and brittle. Extremities usually cold, occasionally hot and congested. Dermatitis, eruptions, and sepsis are common.
   Joints.—Arthropathies. Sudden painless swelling and changes identical with Charcot's joints of tabes occur, but usually in shoulder, elbow, or wrist.
   Spontaneous fractures occur rarely.
3. MOTOR SYMPTOMS.—*Muscular atrophy and paralysis*, of lower [illegible] ne; commonly in sequence—small muscles

of hand (with development of claw-hand), forearm, upper arm, shoulder, as in progressive muscular atrophy. Rarely, commences in shoulder. *Spastic paraplegia* common, though rarely severe (pressure on pyramidal tracts); condition finally resembling amyotrophic lateral sclerosis. *Scoliosis* common, from muscular weakness.

SPHINCTERS and SPECIAL SENSES.—Rarely affected. Cervical sympathetic may be paralysed (small pupil, etc.).

OCCASIONAL VARIATIONS.—*Onset in lower limbs*, i.e., lumbar segments: extension from upper limbs very rare. Rarely, *medulla*, pons, and 4th ventricle affected, with dissociated anæsthesia of face and head, nystagmus, paralysis of cranial nerves, or bulbar paralysis.

Types.—Schlesinger describes five: (1) *Classical type*: usual form. (2) *Motor tracts* mainly affected, resembling amyotrophic lateral sclerosis. (3) *Sensory tracts* mainly affected, resembling hysteria. (4) *Trophic changes* marked, viz., Morvan's disease. (5) *Tabetic type*: posterior columns affected, with tabetic changes in lower and syringomyelic changes in upper limbs.

MORVAN'S DISEASE (*Painless Whitlows*). Marked trophic changes and dissociated anæsthesia in the extremities. Results in necrotic dactylitis. May also be sepsis. Is a type of syringomyelia.

Diagnosis.—Usually simple, from combination of dissociated anæsthesia, trophic changes, atrophic paralysis, and slow progress. Diagnosis from:—

SPINAL HÆMORRHAGE. Onset sudden; improvement follows. Close resemblance of symptoms.

INTRAMEDULLARY TUMOURS. Symptoms more unilateral; progress rapid.

ANÆSTHETIC LEPROSY.—Nerves thickened; loss of tissues.

HYSTERICAL ANÆSTHESIA. Onset sudden; no dissociation; 'glove' or 'stocking' distribution; never an extensor plantar reflex.

TABES (from lumbar syringomyelia).—Argyll Robertson pupil; no muscular atrophy; cerebrospinal fluid and Wassermann reaction.

ERYTHROMELALGIA.—Occasionally simulated; trophic forms.

Dissociated anæsthesia distinguishes from many conditions of wasting, etc.—e.g., progressive muscular atrophy, cervical ribs, pachymeningitis cervicalis.

Course and Prognosis.—*Progress* very slow; may be arrested for many years. Rarely, rapid advance from hæmorrhage into cord. *Death* from bed-sores, sepsis, or intercurrent affections. Prognosis worse in lumbar forms.

Treatment.—Protect from injury to anæsthetic parts.

V. LESIONS OF THE CAUDA EQUINA AND CONUS MEDULLARIS.

Anatomy.—The *conus medullaris* commences, arbitrarily, at upper border of 2nd lumbar vertebra and terminates above its lower border. The *cauda equina* contains the 2nd lumbar and lower nerves until their exit at various levels.

Lesions of the Cauda Equina and Conus Medullaris, *continued.*

Etiology.—

1. FRACTURE of vertebræ or sacrum: may be hæmorrhage. Common cause
2. TUMOURS of roots, membranes, or bone

Rarely:—

3. GUMMATOUS MENINGITIS.

Lesions of Cauda Equina.—

SYMPTOMS.—Vary with site and nerves affected, often *asymmetrical.* General characteristics.

1. ANÆSTHESIA.—'Saddle-shaped' area in gluteal region; perineum; scrotum; urethra.
2. PAIN in nerve areas.
3. PARALYSIS of lower motor neuron type i.e., flaccidity, rapid wasting, reflexes absent, etc. Usually below knee and in buttocks.
4. SPHINCTERS PARALYZED. Bladder and rectum incontinent.
5. SEXUAL POWER LOST.

Lesions of Conus Medullaris.—Resemble lesions of lower portion of cauda equina below 2nd sacral nerves—i.e., reflexes present, and muscles below knee escape—but part of the cauda equina is usually involved simultaneously. The anæsthesia may be 'dissociated', but pain is usually slight.

Diagnosis.—By signs of injury; distribution of sensory and motor changes. From sciatica, by affection of sphincters and bilateral symptoms.

Treatment.—Surgical, unless syphilitic.

VI. HÆMATOMYELIA.

(*Spinal Hæmorrhage*)

Hæmorrhage into the spinal cord is rare.

Varieties.—(1) *Primary*; (2) *Secondary.*

1. PRIMARY HÆMORRHAGE.

ETIOLOGY.—

Age.—All ages, usually 20 to 40 years.

Sex.—Males commonest.

Injury usual cause, especially to neck; neither fracture nor injury to meninges invariable; occasionally in infants during labour.

Hæmophilia, extreme anæmia, violent muscular exertion, are rare causes.

Rarity, compared with cerebral hæmorrhage, due to length and tortuosity of arteries diminishing effects of high blood-pressure.

PATHOLOGY – Hæmorrhage commences in gray matter (from vascularity); may be limited to it, and often unilateral, but extent varies, spreads vertically rather than transversely; commonest in cervical and lumbar regions. Subsequent changes as in other hæmorrhages – viz., scar formation, cavities, and cysts. Surrounding myelitis common.

2. SECONDARY HÆMORRHAGE. — Into areas of *myelitis*, *tumours*, or *syringomyelia*, producing sudden symptoms. *Minute petechial hæmorrhages* occur in tetanus, eclampsia, rabies, and other severe convulsions, and rarely in extreme venous congestion, but symptoms are due to primary disease, and condition is not clinically hæmatomyelia.

Symptoms. –

AT ONSET. *Sudden paralysis*, or occasionally rapid development to a maximum. Consciousness usually retained. Root pains rarely severe. May be marked hyperæsthetic area and pains in back. Initial anæsthesia complete, or dissociated anæsthesia and Brown-Séquard's syndrome from onset.

Symptoms vary with extent and site as in other spinal lesions. Complete transverse lesion frequent, with complete sensory and motor paralysis, absence of reflexes, paralysis of sphincters, and hyperæsthesia in affected segment.

CERVICAL REGION. All limbs affected, also abdominal and thoracic muscles, whence diaphragmatic breathing only.

DORSAL REGION. Arms escape.

LUMBAR REGION. Flaccidity *permanent*, rapid atrophy, sphincters incontinent.

SUBSEQUENT CONDITION. (1) *Trophic paralysis* in affected segments, (2) *Spastic paralysis* in lower segments, with increased reflexes and Babinski's sign, (3) *Dissociated anæsthesia*, (4) *Sphincters paralyzed*, (5) Trophic changes, bedsores and cystitis. Sensory and motor symptoms below lesion may be unilateral (producing Brown-Séquard's syndrome) or bilateral.

Diagnosis. From

MENINGEAL HÆMORRHAGE. – By sudden paralysis, dissociated anæsthesia, and absence of root pains and muscular spasms.

SYRINGOMYELIA. Symptoms often identical, but onset slow and condition progresses, while hæmorrhage improves.

ACUTE MYELITIS. Premonitory symptoms, less rapid onset; pyrexia.

Course and Prognosis.—*Rapid death* common, especially from respiratory paralysis. *Improvement* otherwise considerable, with varying degrees of residual symptoms (*see* 'Subsequent Condition' above). Cystitis or bedsores may be fatal.

Treatment. Absolute rest, many weeks. Ice bag to spine. Laminectomy contra-indicated. Regulate bowels. General and subsequent treatment as in myelitis.

VII. HÆMATORRHACHIS.

(*Meningeal Hæmorrhage.*)

Meningeal hæmorrhage of the cord is very rare.

Varieties.—

1. EXTRADURAL.—Commonest form. From spinal injuries. Rarely, aortic aneurysm.
2. INTRADURAL.—(*a*) Fractured base of skull; (*b*) Ruptured vertebral or basilar aneurysms; (*c*) Spinal injuries. Very rarely from: (*d*) Hæmophilia, purpura, etc.; (*e*) Hæmorrhagic fevers, e.g., small-pox; (*f*) Tetanus, eclampsia, and severe convulsions; (*g*) Extreme venous congestion.

Symptoms.—With moderate degrees, symptoms slight. When severe: (1) Onset sudden; (2) Severe pains in back (meningeal); (3) Severe pain and hyperæsthesia in root areas; (4) Paræsthesia in limbs; (5) Involuntary muscular spasms; rigidity of back. No loss of consciousness, no pyrexia, but often much shock.

Paralysis of limbs, anæsthesia, affection of sphincters, of varying degree, may develop either rapidly or less suddenly, and especially in lower segments, from gravitation of blood. Symptoms vary with extent and level of hæmorrhage.

Diagnosis.—(*See* HÆMATOMYELIA.) From *spinal meningitis*, by sudden onset and absence of pyrexia. Presence of blood in cerebrospinal fluid is of diagnostic importance in fractured base.

Course and Prognosis.—Mortality high, from hæmorrhage, or other injuries, or respiratory paralysis. Prognosis improves after few days, but recovery never complete. May be death from bed sores or cystitis.

Treatment.—Absolute rest, many weeks. Ice-bag to spine. Regulate bowels. Morphia for pain. *Laminectomy* urgently indicated by signs of compression and increasing hæmorrhage. General and subsequent treatment as in myelitis.

VIII. LANDRY'S ACUTE ASCENDING PARALYSIS.

An acute ascending flaccid paralysis commencing in the legs and rapidly extending to the trunk, arms, and diaphragm. No sensory, electrical, sphincter, or mental changes, and no wasting. Reflexes lost.

Note 1.—Few conditions are more in dispute. Some authorities include cases with sensory changes or with various gross pathological changes in cord or nerves. Such forms, undoubtedly occurring, become intermediate with acute polyneuritis, poliomyelitis, and ascending myelitis. They are not included in this description.

The *pathogenesis* also is obscure. Obviously it is an affection of the lower motor neuron. Landry described the march of the paralysis as legs, arms, trunk—i.e., commencing generally from periphery, and suggesting a toxic multiple neuritis ascending the nerves and finally

affecting the cord; a view widely held. But most clinicians consider the order to be legs, trunk, arms—i.e., a process ascending the cord. No unimpeachable organism discovered.

Note 2.—Landry's is not the only form of acute ascending paralysis.

Etiology.—

AGE.—Commonest 20 to 30 years.

SEX.—Males most frequent.

PREDISPOSING FACTORS. Often none, patient previously in good health; occasionally alcoholism, infectious fevers, exposure.

Symptoms. -

ONSET of paralysis sudden. Occasionally *premonitory symptoms* for hours or days or more - e g, paræsthesias, various pains, or weakness. Paralysis commences in lower extremities, at or near periphery, and progressively ascends in order: (1) Legs; (2) Trunk; (3) Arms (commencing at periphery); (4) Diaphragm; and, if surviving, (5) Cranial nerves

DURATION OF PROGRESS. - A few hours to a few days.

PARALYSIS *flaccid*. *All reflexes lost*, deep and superficial. No atrophy or electrical changes. No sensory, sphincter, or mental changes. No pain. Pyrexia absent or slight. Trophic changes not marked. Spleen occasionally recorded as palpable.

VARIATIONS AND ATYPICAL FORMS frequently recorded, with varying degrees of sensory changes, tenderness in nerves, etc (*see Note* 1 *above*). Paralysis may commence in arms, rarely.

Diagnosis. Difficult from other forms of acute and ascending paralysis.

ACUTE MULTIPLE NEURITIS.- Pain, tender muscles and nerve trunks; sensory changes; early wasting; abdominal reflexes present. Mortality low.

ACUTE ASCENDING MYELITIS. Marked sensory changes; sphincters affected; pyrexia. Mortality very high.

ACUTE ANTERIOR POLIOMYELITIS. Severe constitutional symptoms; paralysis rarely complete; no sensory changes, but often pain on movement; rapid atrophy.

Course and Prognosis.- *Death* from respiratory paralysis. Apparently typical cases have recovered, and then often completely. In later stages muscles waste.

Treatment. Maintain general strength. Injections of strychnine. Artificial respiration and inhalation of oxygen when respiration failing. During recovery, treatment as in myelitis.

IX. DISSEMINATED SCLEROSIS.

(*Multiple Sclerosis. Insular Sclerosis.*)

A chronic disease of the nervous system, characterized pathologically by areas of sclerosis irregularly scattered, and clinically by symptoms of spastic paraplegia and by nystagmus, intention tremor, and scanning speech. Not uncommon.

Disseminated Sclerosis, *continued*

Etiology.—

AGE AT ONSET —Commonly 15 to 30 years, very rarely recognized under 12 years

SEX.—Sexes equally affected

PREDISPOSING FACTORS —Doubtful Not *hereditary*, very rarely *familial*. No syphilitic factor

Morbid Anatomy.—*Areas of sclerosis* scattered irregularly through brain and spinal cord, peripheral nerves not exempt *White matter* mainly affected, but frequent in basal ganglia *Outline* of area definite, *shape* irregular, *size* variable (up to a pea, rarely larger) Recent areas soft and translucent, old areas firm

No ascending or descending degeneration from the areas reason doubtful; sometimes ascribed to persistence of axis cylinders, but many are destroyed in later stages

HISTOLOGY OF AREAS OF SCLEROSIS (1) *Myelin sheaths of nerve-fibres absent but axis cylinders present* (in later stages may degenerate), (2) Proliferation of neuroglia

Pathogenesis entirely unknown Most commonly believed that degeneration of myelin sheath is initial change, and due to toxin of autogenous origin

Symptoms.—

SUMMARY (a 'spastic paraplegia' with certain special symptoms)

(1) Weakness and rigidity, especially in lower limbs, (2) Deep reflexes increased, with Babinski's sign, (3) Visual disturbances and optic atrophy, (4) Sphincters affected, together with Charcot's triad, viz (5) Intention tremor, (6) Nystagmus, (7) Scanning speech *

MODES OF ONSET AND INITIAL STAGES -

ONSET may be with *transient* attacks of either. (1) Weakness in limbs and paralyses, commonest; (2) Paræsthesias numbness and tingling, (3) Tremors or ataxia, (4) Visual disturbances

THE INITIAL STAGES (often many years) are characterized by (1) Great variability of symptoms, (2) Transient symptoms and prolonged remissions Attacks occur resembling (*a*) hysteria, (*b*) 'influenza' Thus, e g, paralysis of one leg of sudden onset disappears suddenly or gradually, and after a long interval paralysis of an arm occurs

THE CONDITION PROGRESSES, recovery from attacks becomes less complete, and various characteristic symptoms develop, or are found on examination

CONSIDERATION OF SYMPTOMS IN DETAIL —

1. MOTOR PHENOMENA — Invariably present, may advance to complete disability Lower limbs most affected In

* Characteristic as 5, 6, and 7 may be, the absence of one or even all is far from uncommon, especially in the spinal form, even when the history of the disease can be traced back for many years; and such absence does not negative a diagnosis duly supported by other reasons.

earlier stages, *transient paralyses*. *Spastic paraplegia* gradually develops, with weakness and rigidity, *wasting* uncommon until late stages. *Inco-ordination* variable. *Contractures* occur late. Sudden muscular spasms may be troublesome. *Gait*, as in spastic paraplegia or spastic ataxia: patient drags feet; is unsteady; walks with difficulty, on wide basis.

2. TREMOR.—Characteristic '*intention tremor*', viz.: (*a*) Cessation at rest; (*b*) Occurs during voluntary movement, increasing in severity as movement continues. In *handwriting*, revealed early, marked at end of a sentence. Usually in arms only.

   Theories of Origin: (i) Charcot: Axis cylinders are not properly insulated, and conduction of impulses is irregular. (ii) Erb: Areas of disease in certain sites disturb the mechanism for co-ordination of movement.

   May be absent throughout, or coarse tremors present.

3. OCULAR PHENOMENA.—Important:

   a. Nystagmus.—Bilateral, usually lateral. Present in 50 to 70 per cent.

   b. Primary Optic Atrophy. Mainly pallor of temporal half of discs. In 50 per cent. No retinitis or optic neuritis (*see also* OPTIC ATROPHY). Affects vision, but complete blindness rare. (Cf. TABES.)

   c. Visual Disturbances. Common. Often transient. Transient amblyopia or diplopia common, but obvious ocular palsy rare.

   d. Fields of Vision. May be irregular contraction or central scotoma, often for colour only.

   e. Pupil. Reactions normal.

4. SCANNING SPEECH. Syllables separated and staccato. Characteristic, but often absent. Minor changes common—e.g. monotonous tone.

5. REFLEXES.

   a. Deep reflexes greatly increased: knee, ankle, elbow, wrist, occasionally jaw.

   b. Ankle clonus present, true or 'spurious'.

   c. Extensor planta response, Babinski's sign, very rarely absent. Superficial abdominal reflexes lost early.

6. SPHINCTERS affected. Early: difficult or 'precipitate' micturition. Later incontinence.

7. SENSORY PHENOMENA. Numbness and tingling common. Sensory changes slight or indefinite.

8. PSYCHICAL CHANGES. Patient often emotional. Mental change very rare: may occur late in rare cerebral forms.

Trophic changes in skin, nails, etc., occasionally. Sexual power diminishes. Epileptiform seizures extremely rare.

Types.—

(*a*) SPINAL. Spinal symptoms marked, resembling closely spinal diseases—e.g. (*a*) primary lateral sclerosis i.e., a spastic paraplegia; (*b*) degeneration of postero-lateral columns—i.e.,

Disseminated Sclerosis —Types, *continued*

spastic ataxia (less common) Charcot's triad may be absent throughout

2 CEREBROSPINAL —Both spinal and cerebral symptoms producing classical syndrome

3 CEREBRAL Rare Marked headache, giddiness etc and later psychical changes

ATYPICAL CASES are very common

Diagnosis. Note (1) History of transient paresis etc and marked remissions (2) Presence of characteristic symptoms especially Babinski's sign, and the triad nystagmus intention tremor and scanning speech, but absence of latter does not exclude diagnosis

Diagnosis from

1 HYSTERIA In early stages differentiation difficult and *it may coexist* Note Babinski's sign optic atrophy and Charcot's triad

2 TABES, DEMENTIA PARALYTICA and SYPHILIS OF NERVOUS SYSTEM —Note especially (*a*) Pupil changes (*b*) Wassermann reaction (*c*) Cerebrospinal fluid

3 SUBACUTE COMBINED DEGENERATION Later onset anæmia sensory changes absent knee jerks

Also from

CEREBELLAR DISEASES, INTRACRANIAL TUMOURS FRIEDREICH'S ATAXIA

Course and Prognosis. Commonly there is a long initial stage insidious and deceptive, and a chronic course characterized by remissions Less commonly steadily progressive In final stages exhaustion cystitis bedsores bulbar paralysis or intercurrent disease

DURATION —Longest in *spinal* type up to 20 years or even average life time Shortest in *cerebral* type may be 1 to 2 years

Treatment — Palliative treatment is important Good food fresh air, and exercise, but avoid all fatigue (Worse in cold wet and winter) Massage and passive movements useful electricity contra-indicated

DRUGS —Arsenic beneficial Mercury, iodides and silver nitrate doubtful Strychnine contra indicated

PREGNANCY undoubtedly bad

(Rare Varieties of Sclerosis, usually with dementia *Tuberose sclerosis*, *Diffuse sclerosis* also *Pseudo sclerosis*)

X. HERPES ZOSTER.

(*Zona*)

An acute affection characterized by erythema, vesicles, and pain in the cutaneous area corresponding to one, or rarely two dorsal roots

Etiology.—(1) Idiopathic, (2) In dementia paralytica and tabes, (3) Acute cerebrospinal meningitis, (4) Arsenic poisoning (5) Tumours, caries etc, involving dorsal root ganglion

Pathology. Acute interstitial inflammation of dorsal root ganglia (Head and Campbell) Idiopathic form is probably an acute specific infection Possibly related to chicken pox

Symptoms.

ONSET —Malaise and pain, slight pyrexia

ERUPTION Commences with erythema about third day, then formation of vesicles Commonest on trunk and unilateral Distribution area supplied from a dorsal root (partial or complete) On face common in area of ophthalmic division of trigeminal nerve ('partial fifth')

PAIN Precedes eruption often severe

OCCASIONALLY *Lymphatic glands* enlarged especially in axilla Sensory changes slight and variable rarely paresis

Sequela. *Post herpetic neuralgia* occasionally very severe in old people

Treatment. *Local* simple ointment Pain may need morphia For subsequent neuralgia if severe section of posterior spinal root

CHAPTER CXVII

SYSTEM DISEASES OF THE SPINAL CORD.

I. SPASTIC PARAPLEGIA.

(*Lateral Sclerosis*)

Loss of power and spasticity in the legs, due to lesion or degeneration of upper motor neurons, with absence of affection other tracts Theoretically a bilateral lesion may occur at any site but in adults it is practically always in the cord i e, the lateral pyramidal tracts in children it may be in cord or cortex e g, Little's disease

Occurrence.

1 PRIMARY FORMS (1) *Primary lateral sclerosis* (2) *Hereditary spastic spinal paralysis*, (3) *Erb's syphilitic spinal paralysis*

Note In the two last groups, other tracts are usually involved—e g, posterior columns (postero lateral sclerosis) hence the condition is not a pure lateral sclerosis, *the occurrence of a pure primary lateral sclerosis is still doubtful*

2. SECONDARY FORMS Occurs as *initial* clinical phenomenon in numerous spinal lesions, especially (1) *Disseminated sclerosis* (2) *Transverse myelitis from compression* e g caries, tumour fracture Less common in (3) Amyotrophic lateral sclerosis (4) Syphilitic chronic meningomyelitis. Rarely (5) Dementia paralytica (6) Cerebral tumours in pons, etc Closely simulated in (7) *Hysterical spastic paraplegia.*

Spastic Paraplegia, *continued.*

Upper Motor Neuron Lesions.—*Characteristics* are: (a) Loss of power in muscles supplied; (b) Rigidity; (c) Increased reflexes; (d) Absence of wasting; (e) Absence of sensory changes, electrical changes, and affection of sphincters; (f) Babinski's extensor plantar reflex.

Symptoms of Lateral Sclerosis.—

1. INITIAL SYMPTOMS.—(a) Weakness of legs; easily tired (b) Rigidity and stiffness. May be aching in back.
2. CONDITION DEVELOPED.—(a) *Weakness and rigidity of legs.* (b) *'Spastic gait'*: legs dragged stiffly, due to combination of weakness and rigidity preventing raising. (c) *Spasm of adductors of thighs*: legs close together, may be crossed, separated with difficulty. (d) *Deep reflexes increased*: knee-jerks exaggerated, ankle-clonus present, plantar reflex extensor. (e) No wasting; no sensory, electrical, or sphincter changes. Cramps and spontaneous spasms in muscles often troublesome.

Diagnosis.—From:—

1. DISSEMINATED SCLEROSIS.—Examine for nystagmus, tremors, and alterations in speech.
2. TRANSVERSE MYELITIS.—Sensory changes, signs in back (caries, etc.).
3. AMYOTROPHIC LATERAL SCLEROSIS. Wasting and weakness in upper limbs.
4. HYSTERIA.—Often very difficult. Usually wasting. Ankle-clonus 'spurious' (but this also occurs in early organic lesions). Other signs of hysteria: anæsthesia, fields of vision, etc.
5. SYPHILIS.—Wassermann reaction.

Also examine for signs of lesions of posterior columns (*ataxic paraplegia*).

1. PRIMARY LATERAL SCLEROSIS.

(*Primary Spastic Paraplegia.*)

Symptoms of spastic paraplegia due to primary degeneration of lateral pyramidal tracts in cord, of spontaneous origin, and without affection of other tracts.

The occurrence of such a clinical entity is still doubtful; some chronic cases are on record, but most examples are subsequently proved to be secondary spastic paraplegia, or to have other tracts (e.g., posterior columns) involved; most frequently it is onset of a disseminated sclerosis.

Etiology.—

AGE AT ONSET.—20 to 45 years.
PREDISPOSING CAUSES.—Possibly cold, wet, injury to spine.

Symptoms.—*See above.* Arms may also become affected, and jaw-jerks and arm-jerks be present.

Diagnosis.—Justified only after many years.

Progress.—Slow; may be arrested; or finally patient may be bed-ridden, and death occur from intercurrent disease.

Treatment.—Exercise without fatigue beneficial. For spasms: hot baths, sedatives.

2. HEREDITARY SPASTIC SPINAL PARALYSIS.

(*Familial Spinal Paralysis.*)

A very rare familial disease in which spastic paraplegia develops, usually commencing in early life.

Etiology.—Markedly *familial*, but rarely *hereditary*. Both sexes, boys commoner. Transmitted by either sex. *Onset* usually 4th to 15th years; in a later group between 20 and 30 years.

Morbid Anatomy. Degeneration of lateral pyramidal tracts, mainly in lower segments. Goll's columns may be affected, and direct cerebellar tracts.

Symptoms.—*Initial symptoms*: stiffness of legs and clumsiness in walking. *Progress* very slow. Complete spastic paraplegia develops. Arms may be affected late. Face escapes. Rarely late sensory and sphincter changes. Mental condition normal.

Intermediate and atypical types of familial disease connect with Friedreich's ataxia and hereditary cerebellar ataxia. Diagnosis also from cerebral palsies, caries of spine, and myelitis.

3. ERB'S SYPHILITIC SPINAL PARALYSIS.

Etiology.—A rare syphilitic lesion. *Age at onset*: 20 to 40 years. Commoner in males. Usually two to five years from infection.

Morbid Anatomy.—Incomplete transverse myelitis in lower dorsal region, with secondary degeneration in lateral and posterior columns.

General Characteristics.—

ONSET very gradual: difficulty in walking, retention of urine, pain in back.

SPASTIC PARESIS of legs develops, rarely complete.

REFLEXES increased, but rigidity not extreme. Ankle-clonus and extensor plantar reflex.

SENSORY CHANGES: some girdle pains, paræsthesia, and loss of temperature sense.

SPHINCTERS affected.

IMPROVEMENT WITH TREATMENT: complete recovery rare.

VII. ATAXIC PARAPLEGIA.

(*Postero-lateral Sclerosis.*)

'Ataxic paraplegia' (Gowers) results from combined disease of posterior and lateral columns. Such postero-lateral sclerosis occurs in: (1) *Primary ataxic paraplegia* (possibly—*see below*); (2) *Friedreich's*

Ataxic Paraplegia, *continued*

ataxia; (3) *Spino cerebellar ataxia*; (4) *Subacute combined degeneration of the cord*; (5) *Syphilis* e g, Erb's syphilitic spinal paralysis (*see* SPASTIC PARAPLEGIA) Also in pellagra and ergotism Very rarely in tabes and dementia paralytica

1. PRIMARY ATAXIC PARAPLEGIA.

Occurrence as a primary disease is doubtful, as is primary lateral sclerosis, most cases with an apparently pure syndrome prove to be disseminated sclerosis or subacute combined degeneration

Symptoms.—

WHILE LESION IS CONFINED TO POSTERIOR AND LATERAL COLUMNS – Combination of *ataxia* and *spastic paraplegia*, constituting '*spastic ataxia*'

ONSET in legs, with stiffness, unsteadiness, and rapid fatigue then arms affected

CONDITION DEVELOPED —Combination of (1) Weakness (2) *Rigidity*, (3) *Deep reflexes increased*, viz, knee jerks with ankle clonus and extensor plantar response (Babinski's sign) (4) *Inco ordination*, increased on closing eyes *gait* reeling legs wide apart (5) *Sensation* normal, (6) *Pupils* normal (7) *Sphincters* normal, or affected late

2. FRIEDREICH'S ATAXIA.

(*Hereditary Ataxia*)

A chronic disease commencing in early life characterized pathologically by degeneration of the posterior and lateral columns, and clinically by inco ordination, absence of knee jerks, nystagmus alteration in speech, and deformities

Etiology.—

AGE AT ONSET Usually 2 to 10 years, and up to, but rarely after, puberty.

HEREDITARY FACTORS - Commonly *familial*, but less frequently hereditary, transmitted by either sex Sporadic cases not uncommon Consanguinity and alcohol in parents sometimes recorded. Syphilis, no proved influence

SEX.—About equal.

Morbid Anatomy.—

SPINAL CORD distinctly small—probably congenital Sclerosis of extensive distribution in (1) *Posterior columns*, (2) *Lateral columns*, including (*a*) pyramidal tracts, (*b*) cerebellar tracts, both direct and Gowers', (*c*) Clarke's column (whence direct cerebellar tract arises) also in anterior pyramidal tracts. Lower segments most affected.

Cerebellum, medulla, and pons normal, or very slight changes. Congenital pulmonary stenosis or early myocarditis not uncommon.

Pathogenesis.—Probably congenital early atrophy of nerve tissue (Gowers', 'abiotrophy').

Symptoms.—

ONSET insidious and progress slow, but familial nature may result in early recognition. Commences in legs; arms often soon affected.

EARLIEST SYMPTOMS.—Clumsy and unsteady walking. Also changes in feet and absence of knee-jerks.

CHARACTERISTIC SYMPTOMS.—

1. ATAXIA OR INCO-ORDINATION. In voluntary movements—e.g., picking up pin oscillating movements of limb terminate with a sudden pounce. Romberg's sign either present or absent.
2. GAIT irregular, swaying like a drunkard; feet wide apart, but no stamp as in tabes.
3. TREMORS AND IRREGULAR MOVEMENTS, nodding or swaying, of head and trunk.
4. REFLEXES LOST EARLY: knee-, Achilles-, and arm-jerks. *Extensor* plantar reflex.
5. NYSTAGMUS (lateral) usually early, but not invariably present.
6. SPEECH altered: slow, slurred, explosive, and syllables clipped. Due to ataxia of muscles of speech.
7. DEFORMITIES.—(*a*) *Feet*: Early onset, pes cavus (foot shortened and arch raised) and hammer-toes, great toe hyperextended. (*b*) *Scoliosis*.
8. APPEARANCE.—Dull. Mental powers slow, but otherwise unaffected until late.
9. WEAKNESS OF MUSCLES SLIGHT until later stages, but finally extreme.
10. SENSATION usually normal: may be slight late changes. No pain.
11. PUPILS normal.
12. SPHINCTERS normal.
13. ELECTRICAL REACTIONS usually normal.
14. CRANIAL NERVES unaffected.

Clinical Variations are common: nystagmus may be absent, knee-jerks rarely may be present. *Spino-cerebellar ataxia* has been separated as a special type.

Diagnosis.—Often simple, from early age of onset, familial character, and symptoms. Diagnosis from:—

TABES.—Distinguished therefrom by: tremors, nystagmus, speech, deformities, absence of lightning pains and pupil changes; also negative Wassermann reaction. The very rare juvenile tabes needs care.

DISSEMINATED SCLEROSIS.—Distinguished therefrom by: deformities, absent knee-jerks.

Friedreich's Ataxia—Diagnosis, *continued*

Occasionally confused with —
CHOREA (knee-jerks increased), HUNTINGTON'S CHOREA, PROGRESSIVE NEURAL MUSCULAR ATROPHY.

Course.—Very slowly progressive Walking becomes impossible Later, completely bedridden, but may live many years subsequently

Treatment. —Palliative Massage, electricity, Frenkel's method (*see* TABES)

3. SPINO-CEREBELLAR ATAXIA.

(*Marie's Hereditary Cerebellar Ataxia*)

Etiology. —*Familial* and *hereditary* *Onset* usually 1, to 35 years *Sexes* equal Very rare

Morbid Anatomy. Degeneration of cerebellar tracts, partially of posterior columns, pyramidal tracts escape Cerebellum unaffected (hence Marie's original name superseded)

General Characteristics.—(1) *Inco ordination* of cerebellar type earliest in legs (2) *Gait* reeling (3) *Knee jerks increased*, extensor plantar reflex (4) *Optic atrophy*, with failing sight common, also ocular palsies, ptosis etc (5) *Speech*, as in Friedreich's ataxia (6) *No deformities* (7) Nystagmus not common Slowly progressive but life often long

The condition is closely allied to Friedreich's ataxia and all intermediate grades occur, but it is distinguished from typical forms of the latter by (1) Stronger hereditary factor (2) Later onset (3) Presence of knee jerks (4) Presence of optic atrophy and ocular palsies, (5) No deformities

4. SUBACUTE COMBINED DEGENERATION OF THE SPINAL CORD.

A disease of later life, characterized by severe anæmia, together with sensory and motor symptoms due to combined degeneration of the posterior and lateral columns of the cord

Morbid Anatomy.—

NERVOUS SYSTEM —

SPINAL CORD —Normal in size Degeneration of extensive distribution in (1) *Posterior columns*, (2) *Lateral columns*, including pyramidal and cerebellar tracts Most marked in thoracic region, where little white matter escapes, also extensive in lumbar region Gray matter unaffected

MEDULLA —Degeneration of gracilis and cuneate nuclei

CHANGES IN THE BLOOD Resemble, and may be identical with, pernicious anæmia (anæmia with high colour index) Rarely the colour index is not raised, and change more resembles 'secondary anæmia'.

RELATION OF CHANGES IN BLOOD AND NERVOUS SYSTEM —Requires further elucidation *

(1) Nervous symptoms may precede anæmia latter is said to be absent occasionally

(2) Anæmia may precede nervous symptoms or changes in nervous system only found at autopsy

(3) Both groups may be present on earliest examination

Pathogenesis.– The changes in the blood and nervous system are probably due to the same cause, acting both on the hæmopoietic and nerve tissues

Etiology.–

AGE AT ONSET Rare under 40 years

SEX Commoner in females

PREDISPOSING FACTORS Usually absent occasionally various debilitating conditions Subjects often of poor physique

Symptoms. Combination of (1) *Anæmia* pallor, dyspnœa, palpitations, swelling of legs etc (2) *Sensory and motor symptoms* commencing in legs due to postero lateral sclerosis (a) *Numbness and tingling* (b) *Sensory changes* anæsthesia, pain, (c) *Paraplegia*—usually spastic in early and flaccid in late stage Finally (d) *Wasting*, (e) *Sphincters affected* *Pyrexia* slight

THREE STAGES of the nervous symptoms have been described but variations are common

1 FIRST STAGE —*Slight spastic or ataxic paraplegia* Onset insidious numbness and tingling in legs Then symptoms of spastic ataxia weakness, incoordination spasticity and increased reflexes viz knee jerks increased ankle clonus present and extensor plantar reflex Duration several months

2 SECOND STAGE. *Marked spastic paraplegia* develops rapidly

a *Rapid paralysis* unable to stand Rigidity marked

b *Anæsthesia* commences in legs often of stocking distribution ascends rapidly loss of pain preceding touch in trunk upper limit well defined Girdle pains and lightning pains in legs may occur

c *Reflexes increased*

3 THIRD STAGE *Flaccid paraplegia* supersedes the spasticity

a *Flaccid paralysis* Rapid wasting

b *Deep reflexes absent*, but Babinski's sign usually persists

c *Sphincters* loss of control

d *Anæsthesia* increases

Upper extremities may be affected Œdema usual Mental symptoms frequent terminally

* Part, though only part, of the difficulty arises from the frequent absence of collaboration of a neurologist and hæmatologist resulting in one of the two aspects of the disease being insufficiently investigated or unsatisfactorily recorded

The fact that anæmia follows the onset of the nervous symptoms does not justify its description as 'secondary anæmia', a term which has a special meaning in hæmatology

Subacute Combined Degeneration of the Spinal Cord, *continued.*

Diagnosis.—Characterized by combination of *severe anæmia* with sensory and motor changes. Difficult in early stages. Diagnosis from :—

DISSEMINATED SCLEROSIS.—Distinguished therefrom by : later age, anæmia, anæsthesia, pains, absence of nystagmus.

TABES.—Distinguished therefrom (even in flaccid condition) by : anæmia, anæsthesia, absence of pupil changes, extensor plantar response.

PERIPHERAL MULTIPLE NEURITIS.—Distinguished therefrom by : anæmia, affection of sphincters, extensor plantar response.

TUMOURS INVOLVING CORD.—In these : initial root pains, symptoms asymmetrical.

ACUTE MYELITIS has a more rapid onset.

Course.—*Progressive.* Duration : few months up to two to six years. Final stages rapid. Emaciation and weakness extreme, and death from exhaustion, cystitis, bedsores, and cardiac or respiratory failure.

Treatment.—For *anæmia*, as in pernicious anæmia. General treatment for nervous changes, prevention of bedsores, cystitis, etc.

CHAPTER CXXVII.

MUSCULAR DISEASES.

I. MYELOPATHIC MUSCULAR ATROPHY.

A group of diseases in which *progressive* atrophy of the muscles results from a primary degeneration of the cells of the anterior horns or of the corresponding motor nuclei of the cranial nerves. Hence the lesion is essentially of the lower motor neurons, in some forms the upper motor neurons are also affected. Intermediate forms occur between the various groups.

Types.—The following types are generally recognized :—

1. PROGRESSIVE MUSCULAR ATROPHY.—Sometimes referred to as 'type Aran-Duchenne' or 'Duchenne-Aran'. Degeneration in cells of anterior horns (lower motor neuron). Commonest type.
2. AMYOTROPHIC LATERAL SCLEROSIS.—Degeneration in cells of anterior horns and in pyramidal tracts (lower and upper motor neurons).
3. PROGRESSIVE BULBAR PARALYSIS or GLOSSO-LABIO-LARYNGEAL PARALYSIS.—Degeneration in certain motor cranial nuclei in the medulla. Rare.
4. PROGRESSIVE OPHTHALMOPLEGIA.—Degeneration in oculomotor nuclei. Very rare.

Rare type :—

5. PROGRESSIVE MUSCULAR ATROPHY OF CHILDHOOD (Werdnig-Hoffmann type).

1. PROGRESSIVE MUSCULAR ATROPHY.

(*Chronic Anterior Poliomyelitis*)

A chronic progressive disease of the spinal cord characterized pathologically by degeneration of anterior horn cells, and clinically by wasting and weakness of the related muscles

Etiology.—

AGE Adults, 25 to 40 years

SEX Commoner in males

PREDISPOSING FACTORS Rarely recognizable, is a *primary degeneration* Very rarely commences in injured limbs (? hæmorrhages into cord) or infantile paralysis previously present (Potts) Syphilis, no connection No hereditary or familial factors (*see* WERDNIG HOFFMANN TYPE)

Pathology. Commences usually in *lower portion of cervical enlargement* viz, first dorsal segments

1 ATROPHY AND DEGENERATION OF CELLS OF ANTERIOR HORNS May extend into anterior roots and portions of peripheral nerves As secondary changes, proliferation of neuroglia and occasionally small hæmorrhages

2 ATROPHY OF MUSCLES —Distribution irregular, normal fibres remaining

Pyramidal tracts appear normal microscopically slight changes may be demonstrated by methods of Marchi and Nissl Rarely changes in other tracts

Symptoms. Characterized by *wasting and weakness of muscles*

ONSET insidious usually in small muscles of one hand, commonly right, other hand affected after interval often of months Progress bilateral, but more advanced on one side

1. EARLIEST STAGE —(a) *Thumb muscles* of abduction and apposition affected, (b) Then other *small muscles of hand*, viz, little finger interossei lumbricales

CONDITION OF HAND (a) Wasting of thenar hypothenar and interossei muscles (b) Finger movements difficult

POSITION OF HAND ON DEVELOPMENT (a) 'Main en griffe' or 'claw hand' from unopposed action of long flexors and extensors (b) Thumb rotated outwards, becoming flat with palm (ape hand)

Note Claw hand absent if forearm affected early

2 PROGRESS —

ORDER OF AFFECTION —(a) *Forearm* Flexors before extensors Of flexors fingers before wrists, of extensors, wrists before fingers. (b) *Upper arm and shoulder.* Deltoid early affected then biceps (c) *Serratus magnus*, whence 'winged scapula'. Also rhomboids and lower trapezius

MUSCLES ESCAPING (a) Trapezius, upper portion (even in late stages) (b) Pectoralis major, lower half. (c) Triceps (d) Latissimus dorsi

3 ADVANCED STAGES —*Neck muscles* head hangs forward. *Intercostals and abdominal muscles* respiration diaphragmatic. *Legs* affected late *Face* often escapes.

Progressive Muscular Atrophy—Symptoms, *continued*

GENERAL CHARACTERISTICS —(*See* NEURITIS -LOWER MOTOR NEURON LESIONS)

1. WASTING —Finally becomes extreme and universal, both muscle and fat.
2. FIBRILLARY TREMORS common, marked on striking muscles, occur in muscles previous to obvious changes
3. DEEP REFLEXES diminished or lost, from atrophy of muscles and breaking of reflex arc
4. ELECTRICAL CHANGES 'Partial reaction of degeneration' distinguishes myelopathic from myopathic muscular atrophies
   a. *Nerves* -Response normal in type, but diminished
   b *Muscles* —(i) To faradic current react (through nerves) (ii) To galvanic current (a) sluggish response, (b) A C contraction greater than K C contraction Reactions vary in different parts o same muscle Finally, all response lost
5. SENSATION normal Occasional aching as in over fatigue
6. SPHINCTERS unaffected

Clinical Variations. -Rare (1) *Shoulder* affected first, commences in *deltoid*, lesion in upper portion of cervical enlargement (Lead is a possible factor) (2) *Forearm* first, *no main en griffe* (3) *Legs* first, very rare, commences in peroneal

All intermediate forms occur between progressive muscular atrophy and amyotrophic lateral sclerosis

Diagnosis.—Mainly from conditions causing *wasting of hands*, in most of these pain and sensory changes occur

1. PERIPHERAL NEURITIS AND PERIPHERAL NERVE LESIONS.—Pain, sensory changes, distribution of wasting, causal factors In lead, small muscles rarely affected
2. SYRINGOMYELIA -Sensory changes
3. CERVICAL RIB —Unilateral Sensory symptoms X rays
4. CERVICAL CORD TUMOURS SYPHILIS, CARIES, PACHYMENINGITIS Pain and sensory changes
5. MYOPATHIES.—Resemblance mainly in 'shoulder' type Note (*a*) Onset at earlier age, (*b*) Enlargement of certain muscles, (*c*) No fibrillary tremors, (*d*) No reaction of degeneration, (*e*) Affection of muscles escaping in progressive muscular atrophy. (*See also* MUSCULAR DYSTROPHIES, p 806)
6. AMYOTROPHIC LATERAL SCLEROSIS - (*a*) *Progress* more rapid; (*b*) Often affects muscles in groups (*c*) *Deep reflexes* increased markedly; (*d*) *Spasticity of legs* (*e*) *Bulbar paralysis* common

Course.—Slowly progressive *Death* in five to fifteen or twenty years: usually from diseases or failure of respiration. Development of bulbar paralysis is rare

Treatment.—General hygiene and tonics. Strychnine. Massage. Electricity.

4. AMYOTROPHIC LATERAL SCLEROSIS.

A chronic progressive disease of the spinal cord, characterized pathologically by degeneration of the pyramidal tracts and of the cells of the anterior horns, and clinically by a combination of atrophy and spasticity in the related muscles.

Etiology.—Commoner in females. Otherwise as in progressive muscular atrophy, but is considerably rarer.

Pathology.—Degeneration of both lower and upper motor neurons.

1. CELLS OF ANTERIOR HORNS.—As in progressive muscular atrophy. *Motor nuclei in medulla* (and rarely pons) may also atrophy.
2. PYRAMIDAL TRACTS.—The degeneration extends upwards, and is traced, by methods of Marchi and Nissl, through medulla and pons to cortex.

May *commence* in either of the two sites, or simultaneously in both; commonly in cervical enlargement, occasionally in bulbar nuclei.

Pathologically and clinically, the condition is a combination of progressive muscular atrophy and lateral sclerosis.

Symptoms.—

ONSET insidious. Either: (1) *Wasting and weakness in upper limbs*, as in progressive muscular atrophy, or (2) *Spasticity in lower limbs*; or a combination.

UPPER LIMBS.—*Wasting and weakness.* Order of affection closely as in progressive muscular atrophy—small muscles of hands, forearm, upper arm, and shoulder—but tends to affect groups of muscles, e.g., entire forearm, simultaneously. Similar deformities of hand (claw-hand, ape-hand). Contractures commence.

LOWER LIMBS.—*Spasticity and weakness without wasting* (upper motor neuron type). *Gait* becomes spastic.

ADVANCED STAGES.

1. UPPER LIMBS.—(1) Atrophy. (2) Contractures: flexion of fingers, wrists, and elbow, but generally not extreme.
2. LOWER LIMBS.—Varying degrees of spasticity, flaccidity, and atrophy, but last not to same extent as arms.
3. BULBAR PARALYSIS frequently develops. Speech, tongue, lips, palate, and pharynx affected.

GENERAL CHARACTERISTICS.—

1. WASTING AND ATROPHY, with some spasticity, of upper limbs; SPASTICITY, with some atrophy, of lower limbs. BULBAR PARALYSIS frequent later.
2. FIBRILLARY TREMORS.
3. DEEP REFLEXES greatly and universally increased. Practically sole condition in which *jaw-jerk* occurs. *Ankle-clonus* present. Usually Babinski's sign (not invariably).
4. ELECTRICAL REACTIONS.—Excitability diminished. May be 'partial reaction of degeneration' (*see* PROGRESSIVE MUSCULAR ATROPHY). Finally no response.

Amyotrophic Lateral Sclerosis, *continued*

5. SENSATION normal
6. SPHINCTERS unaffected

Types.—May commence as bulbar paralysis. Other types of onset, and intermediate forms, as in progressive muscular atrophy.

Diagnosis. *Lead poisoning* may, very rarely, produce symptoms resembling the type commencing in the forearms. For diagnosis from other conditions, *see* PROGRESSIVE MUSCULAR ATROPHY.

Course.—Usually fatal in two to four years, especially from development of *bulbar paralysis.*

Special Treatment.—Hot baths, massage, and passive movements to prevent contractures.

3. PROGRESSIVE BULBAR PARALYSIS.

(*Glosso-Labio-Laryngeal Paralysis.*)

A rare disease characterized pathologically by degeneration of motor nuclei of medulla and occasionally of pons, and clinically by atrophy and loss of function in the related muscles.

Not uncommonly occurs in late stages of amyotrophic lateral sclerosis, rarely in progressive muscular atrophy.

Etiology.—As in PROGRESSIVE MUSCULAR ATROPHY.

Pathology.—Primary degeneration of cells in motor nuclei of bulb, most advanced in 11th and 12th nuclei, less so in the nucleus ambiguus (the common motor nucleus of the vago glossopharyngeal nerve); occasionally in motor nuclei of 5th and 7th.

Pyramidal tracts probably always affected to some degree.

Symptoms.—

ONSET and PROGRESS. Gradual. *Tongue, lips, pharynx, and larynx* most affected.

SPEECH affected first, becomes indistinct. Earliest, consonants l, r, n, s, t (linguals), then o, u, p, b, m.

TONGUE.—(1) Weakness in moving and protruding it, (2) Wasting marked, (3) Wrinkling of mucous membrane, (4) Fibrillary contractions.

LIPS become weak (orbicularis oris), with some wasting. Whistling, blowing, etc., impossible.

PARALYSIS OF PALATE.—Voice nasal. Regurgitation of fluids.

SWALLOWING AND MASTICATION affected by (1) Weakness of tongue; (2) Paralysis of palate, (3) Paralysis of pharyngeal muscles. Also by loss of reflex from larynx (food enters glottis).

VOICE affected by (1) Paralysis of palate (nasal tone), (2) Paralysis of adductors of vocal cords (voice feeble, coughing ineffectual); also (3) Paralysis of tongue and lips.

MASSETERS, PTERYGOIDS, and TEMPORAL MUSCLES may be affected.

ELECTRICAL REACTIONS — As in PROGRESSIVE MUSCULAR ATROPHY.

REFLEX from soft palate absent, may be also from larynx (arc interrupted)

SENSATION normal

KNEE JERKS may be increased (pyramidal tracts affected)

ADVANCED STAGE — Characteristic (1) *Mouth* open saliva dribbling (2) *Lower lip* pendulous (3) *Muscles above mouth* unaffected (4) *Tongue* atrophy marked, motionless (5) *Speech* unintelligible (6) *Swallowing* difficult

Course. Progressive and fatal duration about 2 years *Death* from (1) Aspiration pneumonia occasionally suffocation (2) Exhaustion from difficulty in feeding (3) Occasionally, cardiac and respiratory disturbances (vagus nerve)

Treatment. Careful feeding, nasal when necessary

Bulbar Paralysis: General Causes and Diagnosis. — Paralysis of the cranial nerves with motor nuclei in the bulb may result from the following lesions

1 SUPRANUCLEAR — i e 'pseudo bulbar paralysis'

2 NUCLEAR AND INFRANUCLEAR

a '*Acute bulbar paralysis*' (i) Vascular lesions (ii) Post-diphtheritic paralysis and very rarely in other post febrile conditions (iii) Rarely in epidemics of acute poliomyelitis

b *Chronic* (i) Progressive bulbar paralysis (ii) Tumours in medulla very rare (iii) Conditions at the base of the brain i e extramedullary

3 MYASTHENIA GRAVIS

Wassermann reaction should always be tested

PSEUDO BULBAR PARALYSIS Not uncommon Diagnosis difficult Due to bilateral lesions (e g haemorrhage) of tracts between motor cortex and bulbar nuclei most commonly in internal capsule Note (1) Two sides not affected simultaneously (except rarely with lesion just above nuclei) viz history of two attacks of hemiplegia with bulbar symptoms following the second, (2) Paralysis of *upper motor neuron type* i e, no wasting, no electrical changes reflexes present

VASCULAR LESIONS IN MEDULLA (hæmorrhage thrombosis may be syphilitic) Sudden onset, less symmetrical may improve subsequently

POST DIPHTHERITIC PARALYSIS Onset rapid, history of diphtheria or sore throat other nerves affected, short duration, and recovery Very rarely is permanent, but never progressive.

CONDITIONS AT BASE OF BRAIN (meningitis especially syphilitic — tumours, etc) — Not uncommon, but *lesions unilateral.* Usually other symptoms

MYASTHENIA GRAVIS Not (1) Tendency to remissions, (2) 'Myasthenic reaction', (3) No wasting, no reaction of degeneration. No lesions in nervous system (*see* p 811)

Muscular Diseases, *continued*

4. PROGRESSIVE OPHTHALMOPLEGIA.

Very rare. A progressive degeneration of the oculomotor nuclei, corresponding to progressive bulbar paralysis, usually produces ophthalmoplegia externa, very rarely total ophthalmoplegia results externa and interna. Bulbar paralysis may also develop. (See OPHTHALMOPLEGIA, p. 836.)

5. PROGRESSIVE MUSCULAR ATROPHY OF CHILDHOOD.

(*Werdnig Hoffmann Type.*)

A rare disease characterized by *symptoms* resembling progressive muscular atrophy commencing in infancy, with *pathological changes* resembling amyotrophic lateral sclerosis.

Morbid Anatomy. Degeneration of anterior horns and pyramidal tracts. Extensive distribution but not in or above bulb.

General Characteristics.—

1. FAMILIAL disease. Transmitted by either sex.
2. ONSET in infancy, 6 to 9 months. PROGRESS slow.
3. PARESIS AND ATROPHY of muscles, symmetrical, proximal segments of limbs. Largest in thigh, trunk, and pelvis; later, upper limbs and neck. Child unable to walk or stand. Contractures develop.
4. REFLEXES absent. Muscles flaccid. May be fibrillary tremors.
5. ELECTRICAL REACTIONS diminished, or reaction of degeneration.
6. SENSATION normal.

Life rarely exceeds a few years.

II. MYOPATHIC MUSCULAR ATROPHY: THE MUSCULAR DYSTROPHIES.

A group of diseases in which muscular weakness and atrophy result from primary changes in the muscles. In some forms an initial increase in size occurs in certain muscles.

Etiology.

No predisposing factors known except *hereditary and familial.*

Onset in childhood, shortly after birth, or up to puberty; rarely later.

Morbid Anatomy. Muscle fibres atrophied; nuclei increased in number. When 'hypertrophy' present (1) Muscle fibres increased in size but probably not in number. (2) Excess of fat. (3) Increase of connective tissue. The enlargement is mainly (but not entirely) a 'pseudohypertrophy' not due to muscle fibres; the enlarged muscles atrophy later.

Nervous system normal; or slight secondary changes in anterior horns.

Types.—Partly differentiated by presence or absence of 'hypertrophy' of muscles, but some initial enlargement may be present in any type, and intermediate forms occur.

TYPES GENERALLY RECOGNIZED. —

(1) PSEUDOHYPERTROPHIC MUSCULAR PARALYSIS.
(2) ERB'S JUVENILE TYPE.
(3) FACIO-SCAPULO-HUMERAL TYPE (Landouzy-Dejerine type).

RARE AND ATYPICAL FORM—

4. 'DISTAL' TYPE. -Affects fingers, wrists, toes, and ankles; occasionally face.
   Onset: infancy or later. *Diagnosis* from progressive neural muscular atrophy by absence of sensory changes and affection of face.

INTERMEDIATE DISEASES. —

5. PROGRESSIVE NEURAL MUSCULAR ATROPHY. —Allied to both myopathic and myelopathic atrophy.
6. AMYOTONIA CONGENITA.
7. MYOTONIA ATROPHICA. Intermediate type between myopathy and myotonia congenita—i.e., characterized by: (a) Slow relaxation of flexors of hands; (b) Slowly progressive atrophy—sternomastoids, face, anterior thigh muscles, flexors of ankle. *Onset*: 20 to 30 years. *Familial* factor

Diagnosis.—Distinction of *muscular dystrophies* (myopathies) from *myelopathic muscular atrophies*: —

1. ONSET at earlier age.
2. FAMILY AND HEREDITARY FACTORS marked: absent in myelopathies.
3. DISTRIBUTION OF CHANGES. -(a) Affects mainly the *larger* muscles and *proximal* segments of limbs (b) Forearm and hands escape, and deltoid often does so. (c) In some forms, enlargement of certain muscles, viz., calf, infraspinatus, etc. (d) Muscles escaping in myelopathic atrophies are affected viz., trapezius, pectoralis major, latissimus dorsi, triceps (e) No bulbar palsies, larynx never affected.
4. REFLEXES never increased: diminish in relation to wasting.
5. FIBRILLARY TREMORS absent, or occur rarely.
6. ELECTRICAL REACTIONS diminish in general with wasting, but no 'reaction of degeneration'.

Difficulty greatest in rare 'shoulder type' of progressive muscular atrophy.

Diagnosis also from: (1) *Cerebral lesions*: paralysis long precedes atrophy. (2) *Multiple neuritis*: rapid onset, distribution of paralysis, and sensory changes. (3) Progressive neural muscular atrophy.

PSEUDOHYPERTROPHIC MUSCULAR PARALYSIS.

A chronic disease characterized clinically by progressive weakness and atrophy of muscles, with an initial increase in size of certain muscles, and pathologically by absence of primary changes in the nervous system.

Pseudohypertrophic Muscular Paralysis, *continued*

Etiology.—

AGE OF ONSET.—Usually early childhood, 4 to 10 years occasionally as late as puberty, rarely subsequently

SEX.—Males predominate, boys 4 or 5 to girls 1

HEREDITY.—Often familial, the girls usually escaping; tends to be exhibited by males and transmitted by females

Symptoms.—

INITIAL SYMPTOMS. Clumsiness and frequent falls in walking or standing

MUSCLE CHANGES.—

1. DISTRIBUTION OF HYPERTROPHY OF MUSCLES. Most constant (*a*) *Calf* (gastrocnemius and soleus) (*b*) *Infraspinatus* Frequently (*c*) Quadriceps extensor (*d*) Gluteus (*e*) Triceps. Occasionally Deltoid, supraspinatus. Very rarely Masseters, tongue
2. ATROPHY OF MUSCLES. Most constant (*a*) *Latissimus dorsi* (*b*) *Pectoralis major, lower portion* Frequently (*c*) Flexors of knee (*d*) Peroneal (*e*) Erector spinæ and trunk muscles, (*f*) Biceps, (*g*) Teres major
3. MUSCLES ESCAPING (*a*) Face (*b*) Forearm and hand rarely supinator longus wastes

CHARACTERISTIC PHENOMENA

1. ATTITUDE. Stands with feet apart and shoulders thrown back. Marked *lordosis and protuberant abdomen*
2. GAIT waddling, feet wide apart, often lifted high (steppage)
3. RISING FROM GROUND WHEN SUPINE (Gowers' pathognomonic figures). Rolls over on to hands and knees, extends knees with feet apart, moves hands along floor towards span of feet, and then climbs up the legs with a final jerk to the upright position. (Due to weakness of extensors of knees and hips.)
4. SLIPS THROUGH THE HANDS when attempt is made to lift child with hands in axilla. (From absence of axillary folds through atrophy.)

ELECTRICAL REACTIONS diminish quantitatively until finally absent, from atrophy of muscular fibres. *No reaction of degeneration*

SENSATION unchanged

SPHINCTERS unaffected

Course.—Slow progressive atrophy and weakness of muscles including hypertrophied muscles in later stages; finally helpless

DEFORMITIES may develop (1) *Lateral curvature of spine* common, (2) Talipes equinus occasionally, from contraction of gastrocnemius

GENERAL HEALTH fair until terminal stages

DEATH: *usually about puberty*, from exhaustion, or from pulmonary or intercurrent disease.

Treatment. Unsatisfactory General hygiene and tonics *Treatment of muscles* massage, electricity, active and passive movements *Exercise* beneficial Keep from becoming bedridden as long as possible

ERB'S JUVENILE TYPE.

Etiology.—

SEX Boys and girls equally affected (compare previous type)
AGE AT ONSET Commonly second decade in the teens
HEREDITARY factor common

Symptoms.

MUSCLE CHANGES

HYPERTROPHY is never marked may be slight grades

ORDER OF AFFECTION (1) Upper extremity biceps triceps supinator longus and deltoid (especially upper portion) (2) Trunk latissimus dorsi pectoralis major (mainly lower portion) trapezius serratus magnus and rhomboids erector spinæ (3) Thigh and pelvis gluteal flexors and extensors of knee occasionally tibialis anticus

ATROPHY AND WASTING Commences in large muscles about shoulder and upper arm then trunk thigh and pelvis

MUSCLES SPARING (1) *Forearm* (except occasionally supinator longus) (2) *Leg below knee* These contrast with atrophy in proximal segments (3) *Face* (4) Infraspinatus and supraspinatus commonly

CHARACTERISTIC PHENOMENA *Lordosis* common (disappears on sitting) *Attitude gait* and *method of rising from the ground* often as in pseudohypertrophic type

Course. *Progressive atrophy and weakness* but occasionally stationary for some years, duration of life longer than in previous type

FACIO-SCAPULO-HUMERAL TYPE.

(*Type Landouzy-Dejerine*)

Onset in *infancy* with weakness and wasting of *muscles of face* Progress and symptoms otherwise resemble Erb's juvenile type—probably identical *Hereditary* factor common

Symptoms. (1) *Face muscles* affected especially orbicularis oris and palpebralis Characteristics (a) Eyes cannot be closed, (b) Blowing and whistling impossible, (c) Lips everted (d) Smiles with straight lips 'rire en travers' Subsequent involvement of large muscles of (2) Shoulders and upper arm (3) Trunk, (4) Thigh and pelvis (*see* ERB'S TYPE) Hypertrophy of muscles never marked may be slight grades

PROGRESSIVE NEURAL MUSCULAR ATROPHY.

(*Peroneal Muscular Atrophy. Charcot Marie Tooth Type.*)

A chronic disease commencing in early life, characterized clinically by slow muscular atrophy of distal segments of limbs, and pathologically by changes in the nervous system

Progressive Neural Muscular Atrophy, *continued*

A very rare disease, allied to both myopathic and myelopathic atrophies, and possibly to multiple neuritis

Etiology.—

AGE —Onset usually in first decade
SEX —Both sexes but boys 4 or 5 to girls 1
HEREDITARY AND FAMILIAL FACTORS marked Transmitted apparently through females (Herringham)

Morbid Anatomy.— Sclerosis of posterior columns, with atrophy of cells, and of anterior horn, and changes in peripheral nerves and muscles, are most constant, but pathology is still uncertain

Symptoms.—

MUSCLE CHANGES *Atrophy of muscles*, in order (1) *Peronei and small muscles of feet*, whence *talipes equinus* or equinovarus develops (2) Extends up lower extremity until *all muscles wasted below knee* while thigh little affected ('inverted bottle shaped leg') (3) Upper limb affected after interval of several years commences in *small muscles of hand* claw hand develops Often no further progress rarely trunk and thigh muscles affected later
SENSORY CHANGES may occur pains in legs, areas of anæsthesia Muscles not tender
FIBRILLARY TREMORS present
REFLEXES —Ankle jerk absent Knee jerks present
ELECTRICAL REACTION —Varies from quantitative diminution of response to complete reaction of degeneration
SPHINCTERS unaffected

Course:— Very chronic. Often becomes arrested Life not necessarily shortened

General Characteristics.—*Bilateral acquired club foot slow symmetrical atrophy* of *distal* segments, commencing in *early* life Resembles (1) Myopathies in early onset and familial factors, (2) Myelopathies in distal distribution electrical changes, fibrillary tremors, and occurrence of changes in the nervous system Differs from both in sensory changes Differs from *acute poliomyelitis* by slow progress

AMYOTONIA CONGENITA.

(*Myatonia. Oppenheim's Disease*)*

A congenital affection characterized by general flaccidity of the muscles and absence of deep reflexes. Usually noticed at birth or shortly after No familial or hereditary factors Probably a primary disease of muscles classified with muscular dystrophies by some authorities as 'simple atrophic dystrophy

* Oppenheim first described the condition and called it myatonia, a name now generally abandoned owing to its similarity to myotonia (Thomsen's disease).

Symptoms.—(1) *Flaccidity of muscles* extreme. Muscles small, but not atrophied. *Joints are abnormally movable.* (2) *Weakness* extreme, but no paralysis, voluntary control of muscles being present. (3) *Deep reflexes* absent. (4) *Faradic excitability* diminished.

Limbs, especially lower, most affected; face escapes except rarely. No mental, sphincter, or sensory changes, or lesions of nervous system.

Child often unable to walk, but rolls or scrambles over floor.

Course. Tendency to improvement, but death from pulmonary affections common, respiratory muscles being affected.

III. CERTAIN OBSCURE DISEASES.

1. MYASTHENIA GRAVIS.

(*Asthenic Bulbar Paralysis. Erb-Goldflam's Disease.*)

A [illegible] characterized by rapid exhaustion of the voluntary muscles on repetition of movement or stimulation by the faradic current, with recovery on rest. Muscles innervated by the bulb and cranial nerves are specially but not exclusively affected.

Lesion is of *muscular and not of nervous origin.*

Etiology.—

AGE. 20—40 years.

SEX. Sexes affected equally.

HEREDITARY OR NEUROPATHIC FACTOR.—None. Association in several cases with Graves' disease, and in some with congenital malformations, e.g. polydactyly.

Predisposing causes unknown. Has followed infective fevers. May improve during pregnancy, but not invariably.

Morbid Anatomy. Nervous system normal. Main lesions are—

(1) Small round cell infiltrations and serous exudates between muscle fibres and in tissues (Farquhar Buzzard: "lymphorrhagia"); insufficient to affect muscle mechanically.

(2) Thymus proliferation and persistence frequent but not constant. Graves' disease may coexist.

Pathogenesis.—Unknown; possibly faulty metabolism in muscle tissue (cf. MYOTONIA CONGENITA).

Symptoms.

MYASTHENIC PHENOMENON. A movement is performed normally, but on repetition rapidly weakens and becomes impossible; power recovered after rest. More marked towards end of day. Distribution bilateral, but not strictly symmetrical.

MYASTHENIC REACTION.—(1) Strong faradic current: normal contraction, becoming feebler and then ceasing. (2) Galvanic current: reactions unchanged.

DISTRIBUTION OF AFFECTION OF MUSCLES in order of frequency and severity: (1) Muscles supplied by cranial nerves, especially ocular; (2) Neck; (3) Respiration; (4) Limbs and trunk.

Myasthenia Gravis—Symptoms, *continued.*

PROMINENT SYMPTOMS.—

1. OCULAR AFFECTIONS —(*a*) *Bilateral ptosis*: rarely absent (*b*) Orbicularis palpebrarum: slight resistance prevents closure: rarely unaffected (*c*) *Strabismus* and diplopia: finally complete ophthalmoplegia externa Pupil never affected. Coarse nystagmoid movements common.
2. FACIAL MUSCLES, ETC —(*a*) *Power of expression* lost face immobile: whistling, etc, impossible (*b*) Jaw muscles *Mouth open*: saliva drips *Mastication* difficult (*c*) *Palate*: nasal speech, regurgitation of fluids. Articulation impaired.
3. NECK MUSCLES - Inability to hold up head
4. RESPIRATORY MUSCLES Attacks of dyspnœa severe may be fatal.

Later than above:—

LIMBS affected: usually proximal muscles

TRUNK muscles affected

Symptoms exhibited markedly by: (1) Looking up and down repeatedly: ptosis (2) Reading aloud, tires rapidly nasal speech. (3) Tongue put in and out (4) Watch palate while patient says 'Ah' repeatedly movement diminishes rapidly.

SENSATION unchanged Aching, rarely severe pain

ATROPHY OF MUSCLES occasionally in face

Mental condition normal, Sphincters unaffected Knee-jerks weaker on repetition.

Diagnosis and General Characteristics. In advanced cases usually simple: (1) Bilateral ptosis; (2) Facial expression, (3) Nasal speech and open mouth, (4) Rapid exhaustion, (5) No atrophy or sensory changes, (6) Myasthenic reaction (but occasionally absent, and also rarely present in neurasthenia) (7) Remissions

IN HYSTERIA Sensation altered, no myasthenic reaction

IN BULBAR PARALYSIS -Ocular muscles unaffected, no remissions.

Course and Prognosis. Remissions and fluctuations marked Usually fatal in two or more years, from respiratory failure and septic pneumonia

Treatment.—Palliative Feed early in day before exhaustion is marked. Calcium lactate is under trial.

2. MYOTONIA CONGENITA.

(*Thomsen's Disease*)

A very rare affection characterized by a peculiar 'stiffness' on attempting voluntary movements.

Etiology.—

HEREDITARY and FAMILIAL disease

SEX.—Males much commoner than females

Onset noted in childhood from inability to play games

Morbid Anatomy.—

NERVOUS SYSTEM No changes (one autopsy recorded)

MUSCLES Fibres greatly increased in width, transverse striation feeble, nuclei of sarcolemma numerous No increase of connective tissue

Pathogenesis. Unknown Undoubtedly a pathological condition of muscular tissue possibly an error of metabolism In animals similar contractions follow veratria or sodium phosphate in large doses, even after injection of curare (Ringer and Sainsbury) Uncertain whether thickening of muscle fibres is primary or secondary if latter is secondary to original cause and not to peculiarity of contraction

Symptoms.—*On commencing a voluntary movement,* the muscles involved having been at rest the contraction is very slow, and having contracted *relaxation is equally slow On repetition,* stiffness passes off gradually and movement is finally performed at normal rate Well illustrated by shaking hands, gripping being slow and, after closure interval of seven to ten seconds before opening can occur also by attempting to walk after resting

MUSCLES AFFECTED Legs arms and trunk Frequently, mastication and face Eyes rarely

MUSCLES UNAFFECTED Involuntary muscles Respiration deglutition micturition defecation

SENSATIONS and REFLEXES normal

ELECTRICAL REACTIONS *myotic reaction* :—(1) Both to faradic and constant current contraction attains maximum slowly and relaxes slowly on repetition, gradually becomes normal (2) ACC almost equal to KCC

UNAFFECTED by emotion or cold INCREASED by fatigue

CONDITION OF MUSCLES Normal or hypertrophied but force of contraction subnormal

Direct percussion of muscle causes slow contraction.

Treatment. None effective The disease does not shorten life

Atypical Varieties.—

PARAMYOTONIA CONGENITA Condition present in cold weather only Eyes usually affected Non hereditary and other varieties recorded in rare instances

MYOTONIA ATROPHICA Type intermediate between Thomsen's disease and muscular dystrophy (*see* p 807)

3. PARAMYOCLONUS MULTIPLEX.

(*Myoclonus*)

A *rare* affection characterized by sudden shock-like contractions of a single muscle or group of muscles

Etiology.—

SEX — Males most common

AGE Adults

Paramyoclonus Multiplex Etiology, *continued*

PREDISPOSING FACTORS not constant (1) *Shock* may precede onset, (2) *Epilepsy* present in several cases, (3) *Hereditary and family factor* occasional, mainly in cases with epilepsy

Pathogenesis. Chief theories —

(1) AFFECTION OF LOWER MOTOR NEURONS Most probable Symmetrical spasms suggest anterior horn cells

(2) AFFECTION OF CEREBRAL CORTEX Suggested by frequent epilepsy Against this theory is the symmetrical occurrence, and spasm in single muscle or asynergic group

No pathological changes in the nervous system are known

Symptoms. Main characters of spasms are

1. *Limbs* most common Trunk next Face rare
2. *Proximal* muscles commoner than distal viz In arm deltoid and pectoralis major, also biceps triceps and supinator longus In leg quadriceps maximus and adductors of thigh
3. Single muscle, or a portion only may contract *If a group contracts together*, it consists of muscles which are not supplied by single nerve cannot be voluntarily contracted together and have no co ordinated function
4. Limbs usually not moved i e, *no locomotor effect* and spasms not visible until clothes removed Rare exceptions in severe cases
5. *Spasms* usually bilateral and symmetrical one side generally before the other either constantly or in paroxysms Contraction very rapid No constant rhythmicity consecutive contractions do not usually involve the same muscle but may do so Increased by emotion Usually cease in sleep Knee jerks variable

Voluntary movements and co ordination not interfered with

No paresis or muscular atrophy Sensation electrical reactions sphincters unaffected

No psychical or mental disturbance

In the familial form (Unverricht's type, myoclonus epileptica) onset is in childhood epileptic fits occur the contractions interfere with and are increased by voluntary movements and dementia develops

Diagnosis.—From —

1. HUNTINGTON'S CHOREA Dementia develops
2. HYSTERIA —Usually females Stigmata present May closely simulate myoclonus
3. TICS.—Movements purposive

Course.—Chronic No effect on life

Treatment.—Palliative Arsenic and tonics Sedatives may induce habit. hyoscine best Bromides if epilepsy.

CHAPTER CXXVIII.

SYPHILIS OF THE CENTRAL NERVOUS SYSTEM.

Syphilis affects the central nervous system in two forms (1) *Parenchymatous syphilis* tabes dorsalis and dementia paralytica (2) *Interstitial syphilis* lesions of vessels, of membranes and formation of gummata

I. PARENCHYMATOUS SYPHILIS.

1. TABES DORSALIS.

(*Locomotor Ataxia*)

A [illegible] on following syphilis characterized pathologically by degeneration of posterior columns and posterior roots of cord, and clinically by numerous symptoms especially inco ordination pain, and sensory changes loss of deep reflexes, and trophic lesions, due to loss of afferent impulses conveyed in degenerated fibres, also by changes in the pupils and special senses

Etiology.

SEX — Males predominate, about 10 to 1 female.

AGE Commonest 25 to 45 years

SYPHILIS is essential factor presence can be proved in 90 per cent Tabes or dementia paralytica follows syphilis in not more than 5 per cent of cases, probably considerably less (statistics inconclusive).

Morbid Anatomy. —

CHARACTERISTIC CHANGES IN CORD in late st s are —

(1) SCLEROSIS IN POSTERIOR COLUMNS *Macroscopic* Gray and translucent. *Microscopic* (*a*) Degeneration of nerve fibres, (*b*) Increased neuroglial tissue.

(2) ATROPHY OF POSTERIOR NERVE ROOTS

(3) PIA-ARACHNOID THICKENED over dorsal portion of cord

POSTERIOR ROOTS AND COLUMNS OF CORD —

✓ ORIGIN DEGENERATION — The posterior columns contain two sets of fibres (1) *Endogenous* cell bodies outside cord in spinal ganglia fibres form posterior roots, first affected in tabes (2) *Endogenous* cell bodies inside cord [illegible] exogenous fibres not affected until later stages The exogenous fibres—i.e., posterior root—divide into two parts on entering cord (*a*) Lateral small fibres which enter Lissauer's tract (*b*) Internal coarse fibres consisting of (i) Short fibres, at once entering, and ending about cells in posterior horn, (ii) Medium-length fibres which run variable distance in posterior columns just internal to posterior horn then enter horn, or end about cells in Clarke's column and anterior horn; (iii) Long fibres ascending in posterior columns to nuclei in medulla.

ORDER OF DEGENERATION — Commences usually in lumbar region. (1) Medium fibres of internal division degenerate first; hence earliest changes are present in Burdach's column

Tabes Dorsalis—Morbid Anatomy, *continued.*

in lumbar cord. (2) Long and also short fibres next, whence changes in Goll's column. (3) As condition advances, degeneration spreads along fibres outside cord, and finally to cells in posterior root ganglion. Lissauer's tract and collaterals arising in the cord may also degenerate.

Lumbar region (in marked stages).—All posterior columns affected except two small tracts: (i) Cornu-commissural zone, just dorsal to gray commissure; (ii) Oval area of Flechsig, on each side of median fissure.

Cervical region.—Each root on entering pushes towards centre the fibres from lower roots. As cervical roots are rarely affected, the degeneration is here confined to *Goll's columns*, formed by fibres from lumbar region.

ESSENTIAL LESION of tabes is a primary progressive degeneration of posterior root fibres *after* entering cord. Increase of neuroglia and changes in root ganglion are both secondary.

CAUSE OF THE DEGENERATION.—Doubtful. Two main theories: (1) Syphilitic inflammation of pia mater on dorsal surface of cord; supposed to pinch fibres at point where a constriction normally occurs on entering cord. (2) Syphilitic affection or presence of toxins in posterior lymphatic system; fibres lose the neurilemma sheath on entering cord, and hence are susceptible to toxins (Marie).

OTHER SITES OF DEGENERATION.—

1. CRANIAL NERVES. Most commonly *the optic nerve.*
2. PERIPHERAL NERVES.—Rare. Associated with degeneration of anterior horn cells and muscular atrophy, possibly secondary to degeneration of cortex.

Wassermann Reaction.—*See* SYPHILIS, p. 274.

Symptoms—

ONSET.—Most commonly six to fifteen years after syphilis.

THREE STAGES.—(1) Pre-ataxic or incipient; (2) Ataxic; (3) Paralytic.

Symptoms are numerous, and *almost any one may be first to appear or be noted by patient.* A general summary is given, referred to stages, and then the symptoms are considered individually under the various systems. (The functions of the degenerated tracts afford in general an explanation of the symptoms occurring.)

SUMMARY OF SYMPTOMS ACCORDING TO STAGE.—

1. PRE-ATAXIC STAGE.—Any of following may be first noted: (a) *Lightning pains*; (b) *Absence of knee-jerks*, and loss of deep reflexes; (c) *Argyll Robertson pupil.* (The above are the most constant and diagnostic symptoms of tabes.) (*d*) Optic atrophy; (*e*) Difficulty in micturition; (*f*) Romberg's sign. Occasionally: (*g*) Visceral crises; (*h*) Ptosis; (*i*) Paræsthesia. Rarely: Trophic lesions. Impotence not uncommon.

2. ATAXIC STAGE.—In addition to above: (a) Ataxic gait; (b) Inco-ordination of movements. Also: Sensory changes; trophic lesions; hypotonia.

3. PARALYTIC STAGE.—Advanced inco-ordination.

CONSIDERATION OF SYMPTOMS IN DETAIL.—

1. SENSORY SYMPTOMS.—

a. Lightning pains.—Most constant symptom, and often the earliest. Characters: (i) Sharp pains of short duration (few seconds); (ii) Usually in legs; (iii) In bones and muscles rather than joints; (iv) Attacks at irregular intervals, often several weeks; (v) Slight at first, later often intense, but severity has no relation to degree of other symptoms; (vi) May continue in ataxic stage, or may cease (from complete destruction of posterior root).

b. Prolonged pains resembling 'rheumatism' not uncommon.

c. 'Girdle pain'.—Very common. Sense of constriction.

d. Paræsthesia.—Numbness, tingling, formication. Sensation of *walking on cotton-wool* very common.

Objective sensory changes:—

e. Areas of anæsthesia. Usually partial (light touch); area that of spinal segments, especially 4th or 5th dorsal; generally bilateral. Patient often unaware of presence. May be some hyperæsthesia. Ulnar and other nerve trunks may be anæsthetic.

f. Alterations in sense of pain. Various, mainly in legs—e.g.: (i) Delayed conduction; (ii) Felt as touch only; (iii) Loss of localization. *Extremes* of heat and cold as for pain.

g. Impairment of muscle sense.—Position in which limbs are placed is not recognized.

h. Loss of deep sensibility.—Pressure on tendons and bones painless—e.g., squeezing tendo Achillis.

2. ATAXIA.—Due to loss of afferent impulse from muscles, tendons, and joints. Commences in lower limbs. Progress gradual and variable; may advance to *ataxic gait*, and finally to 'paralytic stage', with extreme inco-ordination of all parts, and inability to dress, feed, etc.

Earliest symptoms: Difficulty in equilibrium when washing face or walking in the dark.

Chief phenomena:—

(a) *Romberg's sign* (often early).—Difficulty in standing with heels together, *increased on closing eyes.*

(b) *Ataxic gait.*—Walks bent forward with two sticks. Foot raised high, suddenly thrown out forcibly, and slapped on ground. Knees hyperextended.

(c) *Inco-ordination of movements.*—Tests: Approximating tips of forefingers; touching knee with opposite great toe, etc.

3. HYPOTONIA.—(a) Flaccidity of muscles; (b) Abnormal degree of movements at joints. May occur early. Due, as ataxia, to loss of afferent deep impulses.

Tabes Dorsalis—Symptoms, *continued.*

Paresis.—(*See* OCULAR SYMPTOMS.) Usually slight in limb muscles. Very rarely, paralysis of group of muscles, e.g., peroneal.

4. LOSS OF DEEP REFLEXES, especially *absence of knee-jerk.* - *Early and very important.* Often for years before ataxia. *Loss of ankle-jerk* may precede that of knee-jerk. Test also by 'reinforcement'.

   Superficial reflexes less important: may be increased.

5. PUPIL CHANGES.—(*a*) '*Argyll Robertson pupils*'—i.e., react to accommodation but not to light (loss may be partial, but usually is complete); is present in at least 70 per cent. (*b*) Pupils often small (spinal myosis). *In early stages*, often sluggish reactions: may be inequality.

6. OCULAR SYMPTOMS.—

   a. Optic atrophy.—May be earliest symptom. Usually progresses to total blindness in three to four years. (Primary white atrophy.)

   b. Ptosis.—Unilateral or bilateral. Common early sign.

   c. Paralysis of external ocular muscles. - Often transient—i.e., occasional diplopia. Of all degrees; rarely, total ophthalmoplegia.

7. SPHINCTERS.—Often affected. Earliest sign: *delay and difficulty in micturition.* Retention later, with danger of cystitis and pyelonephritis. Constipation common, in late stages occasionally incontinence (relaxation of sphincter ani).

8. IMPOTENCE.—Usual.

9. VISCERAL CRISES.—Paroxysms of pain in various organs. Usually in early stages. May be first symptom.

   Gastric crisis. - Commonest form. Characters: (*a*) Sudden onset; (*b*) Severe epigastric pain; (*c*) Repeated vomiting, independent of food; (*d*) Hyperæsthesia in epigastrium, with girdle of anæsthesia, not uncommon; pallor, sweating, small pulse, and may be collapse. Duration: up to several days. Attacks often recur every few weeks. Recovery usually rapid.

   Laryngeal crisis.—Not common. Dyspnœa and noisy respiration. May be fatal.

   Numerous rare forms.—Renal. rectal, cardiac (angina), nasal (sneezing), urethral, clitoral.

10. TROPHIC LESIONS.—

   a. Perforating ulcer.—Common site: under great toe. May penetrate to bone. Occasional superficial lesions: *onychia*, herpes, œdema, and local sweating.

   b. Arthropathies (Charcot's joint).—Characters: (i) *Painless rapid swelling of joint*; no signs of inflammation. (ii) *Commonly knee*; occasionally hip, shoulder, ankle, elbow; usually only one joint. (iii) May subside on first attack, but usually recurs; finally a *flail joint.* Occurs at any period, but rarely pre-ataxic.

Pathology: Increased fluid, thickening of synovial membrane; later, hypertrophy and rarefaction of ends of bones, atrophy of ligaments, erosion of cartilage.

Rare trophic lesions:—

c. Brittleness of bones, and fractures.

d. Muscular wasting; associated with atrophy of anterior horns.

II. CRANIAL NERVES AND SPECIAL SENSES.—(*a*) *Ocular* (*see* OCULAR SYMPTOMS). (*b*) *Deafness*: occasionally; from lesion of auditory nerve or labyrinth; may be vertigo. (*c*) *Paralysis of vocal cords*; usually abductors (posterior crico-arytenoids); may also be unilateral atrophy of tongue and palate. Rarely: 5th nerve affected, pain and anæsthesia in area.

Complications.—All rare. *Aneurysm. Cerebral thrombosis* or hæmorrhage from interstitial syphilis of central nervous system. Paraneia and mental changes. Also tabo-paralysis (q.v.).

Rare Variations.—(1) *Cervical tabes*: commencing in cervical roots and upper limbs. (2) *Juvenile tabes*: in congenital syphilis.

Diagnosis.—*Argyll Robertson pupils* with one other symptom is conclusive. Most common group is: (1) Lightning pains; (2) Argyll Robertson pupils; (3) Absence of knee-jerks. Note especially: (4) History of syphilis; (5) *Wassermann reaction* of blood and cerebrospinal fluid (essential in all doubtful cases). Diagnosis from:—

1. MULTIPLE (PERIPHERAL) NEURITIS.—

ALCOHOL, ARSENIC, ETC.—Knee-jerks are absent, but *gait is 'steppage'*, not ataxic; muscles are tender, and Argyll Robertson pupils not present. Occasionally, in alcohol, gait is shuffling and resemblance close (alcoholic pseudo-tabes).

DIPHTHERIA.—Note rapid onset and history of illness. Pupil changes may occur; usually react to light and not to accommodation.

DIABETES.—Perforating ulcer with absent knee-jerks may occur.

2. ORGANIC DISEASE may be simulated by *visceral crises*. *In gastric crises*: area of epigastric hyperæsthesia more extensive than in gastric ulcer, etc. With recurrent gastric attacks in adults, examine pupils and knee-jerks.

3. SYPHILITIC MENINGOMYELITIS.—May closely simulate tabes. Onset at shorter interval after infection, and progress rapid.

4. CEREBELLAR DISEASE.—Ataxia unaffected by closing eyes. Knee-jerks variable: no lightning pains, pupillary, or sensory changes. Headache, vomiting, and optic neuritis common.

Rarely, difficulty from disseminated sclerosis, Friedreich's disease, subacute combined degeneration.

Course and Prognosis.—

COURSE.—Very variable; practically any symptom may be first to appear or to be noticed.

Tabes Dorsalis—Course and Prognosis, *continued*

PRE-ATAXIC STAGE—Duration indefinite, may be many years, or no further advance

OPTIC ATROPHY—When present, *ataxia is very rare.*

LIGHTNING PAINS—Usually diminish in later stages

MENTAL CHANGES WITH PHYSICAL SIGNS OF TABES—When associated, the subsequent course resembles dementia paralytica and not tabes (*See* TABO PARALYSIS)

GENERAL PROGRESS may be (1) Gradual advance (2) Condition becomes stationary even after years, a rapid advance may occur, especially after shock or excesses (3) Rarely, rapid progress in two to three years (young subjects)

RECOVERY never occurs, but there may be improvement

DURATION—Usually ten to fifteen years

PARALYTIC STAGE—Death from tuberculosis, pneumonia cystitis, etc.

Treatment.—

1 GENERAL HYGIENE A quiet regular life exacerbation follows fatigue or excesses Occupation continued if possible

2 DIET—Nutritious Loss of weight common in tabes

3 ANTISYPHILITIC TREATMENT—

INDICATIONS—(a) Onset within five years of infection, (b) No previous course, (c) Syphilitic lesions present, (d) Occurrence or recurrence of tabetic symptoms, e g, lightning pains

METHOD—Salvarsan and mercury inunctions and injections

4 SYMPTOMATIC—

a LIGHTNING PAINS.—Rest Analgesics phenacetin aspirin pyramidon, and finally morphia (frequently unavoidable) Hot baths Counter irritation to spine (blisters) Division of posterior roots as last resort

b GASTRIC CRISES—Rarely controlled except by morphia Mustard plaster or ice to epigastrium

c LARYNGEAL CRISES—Amyl nitrite inhalations

d BLADDER SYMPTOMS—Frequent micturition (two hourly) Catheterize frequently if retention of urine

5. FRENKEL'S SYSTEM OF RE EDUCATION for inco-ordination—Inco-ordination is lessened by repetition of a movement Patient commences by walking along chalked lines at first straight, later zig-zag and complex, also performs simple movements with hands and legs About ten minutes two or three times daily, avoiding fatigue Fresh paths for co-ordination are thus educated

2. DEMENTIA PARALYTICA.

(*General Paralysis of the Insane.*)

An affection following syphilis, characterized pathologically by progressive degeneration of cerebral cortex and meninges, and clinically by mental and physical changes progressing to complete dementia and paralysis.

Etiology.—

AGE —25 to 45 years

SEX —Males predominate

SYPHILIS —As in tabes, causal factor in overwhelming majority Evidence (1) Frequent admission of infection, (2) Wassermann reaction positive in 90 to 99 per cent, (3) Lymphocytosis in cerebrospinal fluid, (4) Immunity to infection by syphilis (5) Juvenile form in congenital syphilis (6) Spirochaetes present in brain

MENTAL STRAIN is a factor i e, syphilization and civilization. Very rarely simulated in sequels of severe head injuries

Morbid Anatomy.—

CHARACTERISTIC CHANGES —

1 DURA MATER thickened and adherent to skull Occasionally hæmorrhagic pachymeningitis

2 PIA ARACHNOID opaque thickened, adherent to cortex, and on removal leaves worm eaten surface

3 CEREBROSPINAL FLUID increased in subarachnoid spaces

4 BRAIN CONVOLUTIONS wasted, especially *frontal and middle lobes*. Mainly atrophy of white matter gray matter reddened from increased vascularity

5 VENTRICLES dilated, fluid increased, ependyma granular Granulations on floor of 4th ventricle

MORBID HISTOLOGY OF BRAIN SUBSTANCE

1 ARTERIOLES —(*a*) Cellular infiltration of perivascular lymph-spaces, (*b*) Proliferation of intima and degeneration of media

2 NEUROGLIA Numerous large spider cells Increase of cells and fibres

3 NERVE ELEMENTS *Betz's cells* (pyramidal cells of cortex) scanty marked chromatolysis Also degeneration and atrophy of cells and fibres

Changes most marked in anterior and frontal lobes but may be present diffusely in basal ganglia, pons, medulla and cerebellum

SPINAL CORD —May be some degeneration of posterior columns (as in tabes) and pyramidal tracts secondary to cortical changes

THEORIES OF ORIGIN May be (1) Primary parenchymatous degeneration of nerve elements with secondary changes in neuroglia and vessels (probable) (2) Primary change in vessels, with secondary changes in neuroglia and nerve elements

Wassermann Reaction.—Blood and cerebrospinal fluid almost invariably positive (*See* SYPHILIS, p 271)

Symptoms.—

ONSET —Insidious Symptoms are *psychical* and *physical*. A *prodromal stage* of psychical changes is often recognizable, of variable duration Physical signs may be present or precede it A *fit* may be earliest phenomenon

PRODROMAL STAGE.—Characterized by early psychical alterations, e.g.: (1) Inattention to business affairs, forgetfulness,

Dementia Paralytica—Symptoms, *continued.*

rapid mental fatigue; (2) Emotional changes—irritability, outbursts of temper, change of affections; (3) Alcoholic and sexual excesses, and unconcealed contraventions of public morals and customs; (4) Senseless expenditures, onset of exaltation and egoism. *Alcoholism* often complicates picture. May grow fat.

ADVANCED STAGE.—*Psychical changes. Delusions of grandeur* and *mental exaltation* marked. Restlessness, sleeplessness, and excitement. Less often, *acute mania.* Occasionally, *neurasthenia* or *melancholia,* replacing or alternating with delusions or delirium. With all types, progressive dementia and paralysis.

PHYSICAL SIGNS.—Become marked in later stages, but changes in pupils, speech, and knee-jerks, and tremors, usually early.

1. PUPILS.—(*a*) *Unequal,* irregular, sluggish reactions—common form; (*b*) Argyll Robertson pupil—less frequent than in tabes. *Optic atrophy* may occur.
2. KNEE-JERKS increased.
3. TONGUE tremulous.
4. SPEECH.—Slow and slurred, syllables often repeated. Changes often early. Tremors of lips and facial muscles during speech.
5. WRITING.—(*a*) Tremulous; (*b*) Omissions of words, etc., from mental change.
6. FACIES of complacent stolidity; often with a childish smile.
7. SEIZURES.—Usually late in disease, but occasionally early. (*a*) *Epileptiform attacks*: either general convulsions, Jacksonian, or like *petit mal.* Automatism may occur. (*b*) *Apoplectiform attacks*: sudden unconsciousness, stertorous respiration, flushing, pyrexia: may be fatal. *Paralyses*—monoplegia, hemiplegia, or aphasia—may follow: are *transient.*
8. PARESIS develops and advances. *Gait* uncertain: often trips on stairs.

SUMMARY.—(1) Mental changes; (2) Characteristic facies; (3) Tremors of tongue; (4) Alterations in speech and writing; (5) Pupil changes; (6) Increased knee-jerks; (7) Seizures; (8) Paralysis; (9) Wassermann reaction positive in blood and cerebrospinal fluid.

Variations of Type.—

1. TABO-PARALYSIS.—Pathogenesis of tabes and dementia paralytica probably identical, one localized mainly in cord, the other in brain. Intermediate forms occur, viz.: (*a*) Onset as in tabes; later progress as in dementia paralytica. (*b*) Mental changes at onset; later progress as in tabes (rarer than preceding type). (*c*) Symptoms combined from onset—typical 'tabo-paralysis'. *Also note*: (*d*) Optic atrophy in tabes commonly followed by mental changes and not by ataxia.
2. PROGRESSIVE DEMENTIA WITHOUT EXALTATION.
3. NEURASTHENIC OR MELANCHOLIC TYPE.
4. CONVULSIVE TYPE.—Numerous seizures with rapid paresis and dementia.

'JUVENILE DEMENTIA PARALYTICA'.—In congenital syphilis: commoner than tabes. Onset 14 to 18 years.

Diagnosis.—Early diagnosis very difficult: suggested by psychical changes, and proved by Wassermann reaction. Diagnosis from:—

1. CEREBRAL SYPHILIS.—May simulate closely. Note: (a) Onset earlier after infection, one or two to five years; (b) Progress more rapid; (c) Delusions of grandeur and exaltation rare; (d) Paralyses of cranial nerves, etc., and convulsive seizures more common; (e) Improvement under treatment.
2. INTRACRANIAL TUMOURS (especially in frontal lobe).—Simulation in rare cases. Symptoms of increased intracranial pressure present, and syphilitic reactions negative.
3. MELANCHOLIA AND NEURASTHENIA.
4. LEAD ENCEPHALOPATHY.—Resemblance rare.
5. SEVERE HEAD INJURIES.—Resemblance in rare cases.

SPECIAL DIAGNOSIS.—(1) *Wassermann reaction*: almost invariably positive in both blood serum and cerebrospinal fluid. (2) *Cerebrospinal fluid*. (a) Lymphocytosis; (b) Albumin present.

Course.—*Onset* insidious. *Gradual progress* until paralytic, demented, incontinent, and bedridden; bedsores common. *Duration* two to five years; rapid if seizures numerous. *Remissions* common; for several months may resume business. *Death* from exhaustion or intercurrent diseases.

Treatment.—Quiet life. With dementia or mental changes, asylum and certification advisable, *preferably early*. Care necessary to prevent bedsores and cystitis. Convulsive seizures: bromides. Mental excitement: sulphonal, or injections of hyoscine (gr. $\frac{1}{100}$). *Antisyphilitic treatment* value much less than in tabes, and may aggravate condition.

II. INTERSTITIAL SYPHILIS.*

Pathology.—Three groups of lesions:—

1. ARTERITIS.—Syphilitic endarteritis obliterans—viz. proliferation or intima, with thickening of media and adventitia; gummatous changes and perivascular infiltration often co-exist (*see* DISEASES OF ARTERIES).

   SPECIAL SITES. Middle cerebral artery and branches, basilar and vertebral, internal carotid. Lenticulo-striate arteries commonest site.

   SYMPTOMS.—*Thrombosis* may result, whence rapid or sudden *aphasia, hemiplegia*, or local paralysis, depending on site: either transient or permanent.
2. MENINGITIS.—(a) Dura mater (*see* PACHYMENINGITIS HÆMORRHAGICA); (b) Pia-arachnoid or leptomeningitis. The latter is common form of 'syphilitic meningitis': often associated with changes in vessels and gummata. Most common type is a diffuse gummatous meningitis at the base (basal meningitis), a gelatinous formation enclosing all the structures.

* *See* Judson Bury, *Diseases of the Nervous System*, Manchester University Publications.

Interstitial Syphilis—Pathology, *continued.*

3. GUMMATA.—May be: (a) Local growths, acting as other tumours; (b) Diffuse: commonly commencing in pia-arachnoid (gummatous meningitis), and tending to spread along vessels into brain tissue.

SPECIAL SITES.—Optic chiasma, interpeduncular space, cranial nerves.

Other forms are: (a) Periostitis or osteitis: in skull not uncommon, in vertebræ very rare. (b) Gumma arising in dura mater: rare.

General Characteristics.—

ONSET.—Comparatively shortly after infection; usually two to five years, may be earlier.

PATHOLOGY.—(1) The various types of lesion frequently co-exist—i.e., meningitis, arteritis, and diffuse gummatous conditions; (2) They occur at multiple sites.

SYMPTOMS, in accordance with above distribution, have following general characters: (1) Multiple, in various combinations, irregular, and asymmetrical; (2) Often incomplete and transient—disappear, reappear, and others occur. Syphilis is suggested by a combination and sequence of symptoms inexplicable by a single lesion, and by the variability and irregularity of their occurrence; the individual symptoms are identical with those due to other causes. The pathological lesions are usually more extensive than the symptoms suggest.

General Diagnosis.—Depends upon: (1) Distribution and variability of symptoms, especially cranial nerve lesions; (2) History of syphilis; (3) Wassermann reaction; (4) Examination of cerebrospinal fluid; (5) Results of treatment.

Clinical Groups.—The most important are: (1) *Intracranial or cerebral syphilis*: (*a*) Meningitis, basal and cortical; (*b*) Thrombosis; (*c*) Gumma. (2) *Spinal syphilis*: (*a*) Chronic meningomyelitis; (*b*) Acute myelitis. Numerous rare clinical types occur. (3) *Cerebrospinal syphilis*: a basal meningitis may spread into the cervical cord, or be associated with lesions in the lumbar zone. Such, and other, combinations constitute cerebrospinal syphilis.

CEREBRAL OR INTRACRANIAL SYPHILIS.

Onset.—Chronic: rarely acute.

Early and Prodromal Symptoms (absence of all is rare).—

HEADACHE.—Severe: *worse at night.* May be local, with tenderness on pressure.

INSOMNIA.—Often due to headache.

MENTAL APATHY AND ATTACKS OF SOMNOLENCE.

VERTIGO, DEFECTIVE MEMORY, IRRITABILITY may be present.

Varieties.—

CORTICAL MENINGITIS.—

LOCAL SYMPTOMS, depending on site of lesion. Note :—

1. *Headache.*—Often frontal or parietal, local tenderness on pressure.
2. *Mental Symptoms* common : forgetfulness, indistinct speech, dementia.
3. *Convulsions*, when motor cortex affected, resembling epilepsy except for sequel of :
4. *Aphasia, monoplegia, hemiplegia*, etc. ; often transient.

BASAL MENINGITIS.—

CHIEF PHENOMENA.—

1. *Headache* : severe, especially nocturnal. Vertigo, vomiting attacks common.
2. *Psychical Changes* frequent : somnolence, stupor, excitement, or delusions.
3. *Epileptiform Attacks* may occur : of all varieties hemiplegia may follow.
4. *Cranial Nerve Paralyses*, especially second, third, and sixth. Very important. Note : (*a*) Optic nerve : may be : (i) Optic neuritis, with subsequent atrophy and blindness ; (ii) Hemianopia of varying extent, from involvement of chiasma or tract. (*b*) Third nerve : very common ; affection usually partial—e.g., ptosis, paralysis of single muscles, pupil changes. (*c*) Sixth nerve : very common ; usually unilateral ; *diplopia* results. (*d*) Fourth nerve : less frequent. Rarely, complete ophthalmoplegia. Less commonly, but not infrequent : (*e*) Seventh and eighth nerves : usually together. (*f*) Fifth nerve : usually sensory portion. (*g*) Tenth, eleventh, and twelfth nerves ; when meningitis is spreading towards cord ; unilateral paralysis of tongue, palate, vocal cords ; also vagus disturbances. A unilateral *bulbar paralysis* suggests syphilis.

COURSE.—Usually remissions and relapses over several years. May be fatal within a year, and even during course of treatment.

ARTERIAL THROMBOSIS, GUMMA.—*See* PATHOLOGY, p. 823.

PSEUDO-GENERAL PARALYSIS, SYPHILITIC DEMENTIA.—Symptoms in diffuse cerebral lesions may closely resemble dementia paralytica (q.v.). Cranial nerve paralyses and other indications of widespread lesions usually appear.

SPINAL SYPHILIS.

Onset.—Usually within five years of infection : may be during period of eruption. Chronic forms sometimes after much longer interval.

Varieties.—

CHRONIC MENINGOMYELITIS.—Commonest variety of spinal syphilis. Lesion usually in dorsal region. Duration of progress variable : few weeks to months.

Spinal Syphilis—Varieties, *continued.*

SYMPTOMS.—

Initial symptoms, due to meningitis: pain in back, especially nocturnal; pain in root areas, and paræsthesias.

On extension to cord, symptoms of incomplete transverse myelitis :—

Sphincters early affected: retention of urine. *Impotence* not infrequent.

Paraplegia, partial or complete; rapidity of onset variable.

Sensory changes variable: partial anæsthesias, often 'dissociated'.

Deep reflexes usually increased. Plantar reflex often extensor.

Signs of cerebral syphilis (cranial nerve paralyses, etc.) often present or precede.

COURSE.—Recovery may be partial, rarely complete. May become stationary. Death from bedsores, cystitis and pyelonephritis, intercurrent diseases; in rapid forms in six to twelve months.

ACUTE MYELITIS.—Onset, six months to five or more years after infection. Due to vascular disease and thrombosis resulting in degeneration and softening.

ONSET.—(1) May be rapid: few hours or days. (2) There may be premonitory symptoms: headache, vertigo, diplopia, difficulty in micturition, etc., due to cerebral changes. Root symptoms (radiating pains, paræsthesia, etc.) are absent, as meninges are unaffected.

SYMPTOMS of transverse myelitis develop :—

Paraplegia, usually spastic, but flaccid in complete transverse myelitis.

Deep reflexes increased or diminished.

Sphincters usually paralyzed.

Sensory changes: commonly anæsthesia up to, and hyperæsthesia at, level of lesion; generally partial.

COURSE.—(1) Flaccid type: may become spastic in few days, but if persistent, generally rapidly fatal. (2) Spastic type commonly improves, often markedly, but complete recovery rare. *Bedsores, cystitis,* and pyelonephritis common.

VARIOUS CLINICAL TYPES.—Practically every disease of the spinal cord is simulated occasionally by spinal syphilis, owing to meningitis, vascular lesions, and resulting degenerations in various sites, e.g. :—

1. SYPHILITIC PSEUDO-TABES.—Rare. Onset earlier and progress more rapid than tabes, and improvement under treatment.
2. DISSEMINATED SCLEROSIS.—Nystagmus and intention tremor absent.
3. SYRINGOMYELIA.
4. ERB'S SYPHILITIC SPINAL PARALYSIS (q.v.).

VARIOUS PATHOLOGICAL LESIONS.—

MENINGITIS. —Gummatous mass enclosing cord, usually small area. Symptoms as in non-syphilitic meningitis, but cranial nerve paralyses, etc. (cerebrospinal syphilis) often present.

VERTEBRÆ. —Rarely affected. periostitis, osteitis, gumma. Symptoms as in tumour or caries.

ISOLATED GUMMA OF SPINAL CORD AND MEMBRANES.—Very rare.

Treatment of Interstitial Syphilis of the Central Nervous System.—*See* SYPHILIS.

CHAPTER CXXIX.

DISEASES OF THE CRANIAL NERVES.

I. OLFACTORY NERVE.

Lesions may occur at any site from nasal mucous membrane (especially anosmia) to cerebral centres in hippocampus and uncinate gyrus.

Anosmia (loss of sense of smell) —Causes —

(1) AFFECTIONS OF THE OLFACTORY MUCOUS MEMBRANE —Common in chronic nasal catarrh, polypi, etc. Transient in acute catarrh, and after strong odours.

(2) LESIONS OF THE BULB OR TRACT. —E.g., head injuries, tumours, meningitis, caries of bone.

Parosmia (perversion of sense of smell). —Occurs in. (1) Insanity — not uncommon; (2) Aura of epilepsy—rarely; (3) Hysteria Rarely in head injuries, tumours of hippocampus.

Hyperosmia (increased sensitiveness). —Occasionally in hysteria; usually with parosmia.

Tests of Smell.—Essential oils, e.g., cloves, peppermint. Ammonia stimulates the *fifth* nerve.

II. OPTIC NERVE AND TRACT.

1. OPTIC NEURITIS AND RETINITIS.

Optic Neuritis, or Papillitis.—

GENERAL APPEARANCE.—

DISC.—(1) *Pink colour* (from dilatation of small vessels); (2) *Edges blurred*; (3) *Disc swollen*; (4) *Physiological cup filled in*; (5) *Vessels hidden in places* by exudation; (6) *Vessels appear 'kinked'* at edge of disc (from passage over swelling).

VEINS IN RETINA distended and tortuous.

ARTERIES small.

Optic Neuritis, *continued.*

Notes.—*In early stage*: disc pink and edges blurred and striated. *Swelling of disc*: on passing from retina to disc in examination, + lenses are necessary. (*N.B.*—3 D = 1 mm.)

VISION.—Often unimpaired.

SEQUEL.—If slight, may recover. If severe, secondary optic atrophy may develop, with impaired vision.

'Optic neuritis' is probably always due to rise of intracranial pressure causing obstruction immediately proximal to disc (papilla); hence 'papillitis' or papillœdema is correcter term.

Retinitis.—

GENERAL APPEARANCE.—

1. HÆMORRHAGES.—In course of vessels. Colour bright-red to black, depending on age. Shape and size vary; may be flame-shaped.
2. WHITE PATCHES.—Two types: (*a*) Glistening white spots, arranged as '*stellate figure*' round macula, or fan-shaped, from crinkling of the retina due to œdema (Marcus Gunn). (*b*) 'Woolly' white patches scattered over retina; origin may be (i) fibrinous exudation, (ii) fatty degeneration in retina, (iii) clumps of leucocytes, (iv) sclerosis of retina.

Note.—'*Stellate figure*' occurs mainly in (1) albuminuria, (2) syphilis.

Diffuse cloudiness of retina common, from serous effusion.

Causes and Varieties of Optic Neuritis and Retinitis.—Optic neuritis and retinitis may occur together or separately. The medical causes of the two conditions are given below, and the differences noted.

1. INTRACRANIAL DISEASE.—Increased intracranial pressure causes *optic neuritis. Retinitis absent.*

   a. INTRACRANIAL TUMOURS.—Produce 'choked disc' (great swelling of nerve head). *Frequency* varies with site of tumour: in cerebrum, usually present; in corpora quadrigemina, always; in cerebellum, in 90 per cent; in pons, rarely; in medulla, very rarely; hence absence does not negative tumour. Intensity varies with rapidity of growth rather than size of tumour. *Onset* may be unilateral, and most advanced on side of tumour, but distinction is difficult. *Subsidence* after decompression often rapid.

   b. CEREBRAL ABSCESS.—Often absent.

   c. MENINGITIS.—Most frequent in basal meningitis—e.g., syphilitic. In cerebrospinal meningitis not common. In tuberculous meningitis, duration rarely sufficient to become severe. *Choroidal tubercles* are distinguished from *retinitis* by: (i) Size; (ii) Not crossed by choroidal vessels; (iii) Indistinct edges; (iv) Absence of pigment (distinguishes from choroiditis).

   d. SYPHILIS OF THE NERVOUS SYSTEM.—Retinitis and choroiditis may also occur.

   e. HYDROCEPHALUS.

2. TOXIC CONDITIONS.—

a. ALBUMINURIC RETINITIS.—*Occurrence*: especially chronic interstitial nephritis; also nephritis of pregnancy. *Variations*: optic neuritis often present, may predominate; or retinal hæmorrhages may be most marked. *Common characteristics*: (i) Stellate or fan-shaped figure marked; (ii) Flame-shaped hæmorrhages; (iii) Arteries small, with distinct white line, rigid, and constrict veins where crossing; veins engorged. *Vision*: often definitely affected. (Blindness in albuminuria may be uræmic, with no fundus changes). *Sequelæ*: Severe optic neuritis may progress to atrophy; in pregnancy may subside, even when severe.

b. DIABETIC RETINITIS.—Usually elderly patients with chronic diabetes. *Note*: (i) No stellate figure; (ii) Optic neuritis absent (or rare); (iii) Round hæmorrhages and scattered white patches numerous; hæmorrhage into vitreous may cause permanent opacities. Diagnosis from albuminuric retinitis usually uncertain.

Other ocular conditions in diabetes: Cataract, toxic amblyopia, retrobulbar neuritis and its sequelæ.

3. BLOOD DISEASES.—*Retinitis* optic neuritis very rare.

a. LEUKÆMIA.—Characters: (i) Eye ground pale (not invariable); (ii) Hæmorrhages numerous, and yellow patches; (iii) Vessels dilated; (iv) No stellate figure.

b. PERNICIOUS ANÆMIA.—Characters: (i) Disc and eye-ground very pale; (ii) Hæmorrhages with white centre; (iii) Vessels distended, especially veins; (iv) White spots scanty, no stellate figure.

In simple anæmia.—Blindness occasionally occurs after large hæmorrhage, usually after few days' interval; generally no changes in fundus; rarely permanent. In chronic anæmia, very rarely, optic neuritis occurs; improves with treatment of cause.

4. SPINAL DISEASES.—Very rare. Optic neuritis recorded occasionally in myelitis (? toxic), and cervical caries and tumours (? interference with cerebrospinal fluid).

5. RETROBULBAR NEURITIS.—Optic neuritis may follow.

6. VARIOUS DISEASES OF THE RETINA.

7. VARIOUS RARE CAUSES OF OPTIC NEURITIS.—Influenza, scarlet fever, pyæmia, lead, alcohol, and other causes of multiple neuritis.

In *hypermetropia* congestion of the discs occurs, simulating early optic neuritis.

2. OPTIC ATROPHY.

May be *primary*, or *secondary* to preceding optic neuritis.

Primary Optic Atrophy:—

APPEARANCE.—(1) Edges of disc sharply defined; (2) Physiological cup deep and *lamina cribrosa visible*; (3) Arteries small or normal; (4) Colour of disc white or grayish.

Optic Atrophy, *continued.*

CAUSES.—(1) *Tabes*: Disc gray (progresses to blindness). (2) Dementia paralytica. (3) *Disseminated sclerosis*: Disc white (never complete blindness). (4) *Excesses*: Alcohol, tobacco, and sexual, especially together. (5) Certain drugs, especially atoxyl, methyl alcohol, rarely lead. Occasional forms: (5) *Hereditary* (Leber's disease); exhibited by males, and transmitted by females; rare. (6) Trauma to the temples. (7) Sudden anæmia from loss of blood. *Retrobulbar neuritis* may result in primary, or less often secondary, atrophy.

Secondary Optic Atrophy.—

APPEARANCE: (1) Edges of disc blurred and irregular; (2) Physiological cup filled in; (3) Arteries small, often white line at side, from previous disease; (4) Colour of disc dead white.

CAUSES as in optic neuritis.

Symptoms.—Vision always affected: (1) Vision impaired; (2) Field of vision diminished; (3) Colour vision fails, red and green first.

3. RETROBULBAR NEURITIS.

Lesion of the optic nerve proximal to nerve head (the 'disc' or papilla).

1. ACUTE FORM.—
   OCCURRENCE.—In *disseminated sclerosis*, also methyl alcohol poisoning, rarely in influenza.
   SYMPTOMS.—Rapid loss of sight, one or both eyes, with central scotoma, pain in eyes.
   PROGRESS to optic atrophy, which may be primary or secondary, depending on whether inflammation reaches papilla and produces optic neuritis. *Vision* permanently affected, but complete blindness rare.
2. CHRONIC FORM.—
   OCCURRENCE.—In excess of tobacco and alcohol, especially together; rarely in diabetes, lead, and possibly gout.
   SYMPTOMS.—Bilateral; impaired vision; central scotoma for colour.
   APPEARANCE.—(*a*) Early stage, hyperæmia of disc; (*b*) Later stage, pallor of temporal half of disc.
   PROGRESS to optic atrophy unusual; recovery common, partial or complete.

Disturbances of Vision without Changes in the Fundus.—Some are functional; others are due to retrobulbar neuritis. Examples:—

1. TOXIC AMAUROSIS.—Especially in *uræmia*, with or without convulsions. Other conditions: diabetes, loss of blood, lead.
2. TOBACCO AMBLYOPIA.—As in retrobulbar neuritis.
3. HYSTERIA.—Helical restriction of fields of vision, and changes in colour fields, etc. Also other functional conditions.
4. NYCTALOPIA (night blindness).
5. CONGENITAL COLOUR BLINDNESS.

4. AFFECTIONS OF OPTIC CHIASMA.

Cause of Lesions.—*Tumours of pituitary gland*, cerebral syphilis, rarely hydrocephalus.

Symptom.—*Heteronymous hemianopia.* Distribution depends on site of lesion :—

1. CENTRAL PORTION OF CHIASMA AFFECTED (most common).—Bitemporal hemianopia results (fibres affected from nasal half of each retina). Extent progresses to total blindness with increase of lesion.
2. OUTER SIDE OF CHIASMA AFFECTED (very rare). —Nasal hemianopia results. extremely rarely bilateral (tabes, calcification of internal carotid arteries).

5. AFFECTIONS OF OPTIC TRACT.

Cause of Lesions.—Tumours from base of brain, rarely hæmorrhage

Symptom (in unilateral lesion). — (Bilateral) homonymous or lateral hemianopia, partial decussation of fibres having taken place at chiasma Thus, a lesion of the *right* tract causes inexcitability of right half of each retina, whence blindness on the *left* side of body.

6. AFFECTIONS BETWEEN THE OPTIC TRACT AND THE VISUAL CENTRES.

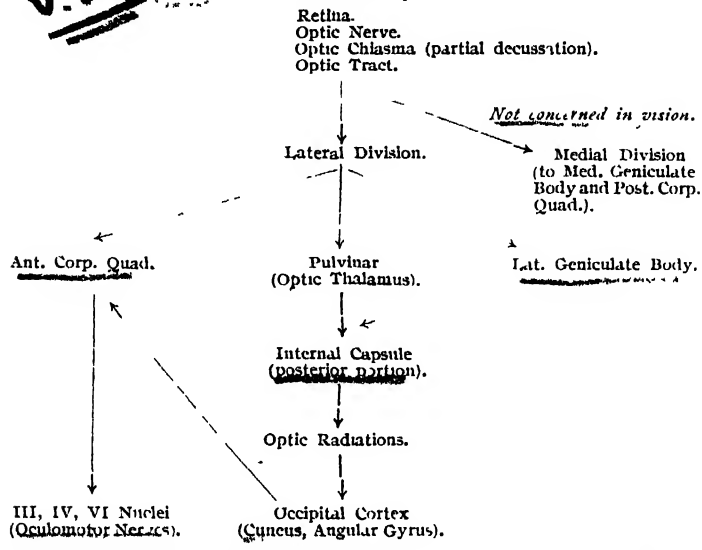
The Visual Paths.—The optic tract crosses the crus or cerebral peduncle, and at the posterior end of the optic thalamus divides into two parts :—

1. MEDIAL DIVISION—Contains fibres from the 'inferior (Gudden's) commissure', passes to the median geniculate body and posterior corpus quadrigeminum Is not connected with retina or concerned with vision.
2. LATERAL DIVISION.—Sends fibres to (*a*) the lateral geniculate body, (*b*) pulvinar (optic thalamus), (*c*) anterior corpus quadrigeminum. From the first two, fibres run in the posterior portion of the internal capsule and the optic radiations to the occipital cortex. *The anterior corpora quadrigemina* are connected by fibres with the nuclei of the third, fourth, and sixth nerves, thus connecting the retina with the nerves controlling eye movements, fibres also run from the occipital cortex to the anterior corpora quadrigemina. The portions of the cerebral cortex concerned with vision are (*a*) the *cuneus* and lingual gyrus, and (*b*) higher centres in the *angular and supramarginal gyrus*.

The lateral geniculate body is apparently connected with the macula lutea, and lesions always affect vision.

Affections between the Optic Tract and the Visual Centres, *continued.*

CHART OF VISUAL PATHS.



Symptoms and Localization.—*Lesions of the visual path between optic chiasma and cerebral cortex at any point produce lateral hemianopia.* Localization must rely on :—

1. The presence of symptoms due to simultaneous lesions of other fibres.
2. The partial character of the hemianopia, as the fibres separate. Behind the lateral geniculate body it is rarely complete.
3. Wernicke's hemiopic pupillary reaction. The pupil reflex takes place through the arc—retina; optic nerve, chiasma, and tract, to anterior corpus quadrigeminum, hence by Meynert's fibres to third nucleus, and by third nerve to ciliary ganglion, ciliary nerves, and iris; the centre is probably in the ciliary ganglion. For the test: A beam of light is directed on the non-functioning portion of the retina; if the pupil contracts, the lesion must be beyond the arc.

SITES OF LESION AND LOCALIZING SYMPTOMS.—Lateral hemianopia is present in all cases.

1. Optic Tract to Lateral Geniculate Body.—Pupil reflex absent.
2. Internal Capsule (posterior portion).—*Hemianæsthesia* not uncommon (sensory fibres close), or hemiplegia (e.g., left

internal capsule lesion produces right hemiplegia and right lateral hemianopia). Pupil reflex normal.

3. OPTIC RADIATIONS.—Hemianopia less complete. Pupil reflex normal.

4. CUNEUS AND LINGUAL GYRUS.—Hemianopia still less complete: may be quadrantic. Bilateral disease causes total blindness. Pupil reflex normal.

5. ANGULAR GYRUS.—Results usually not in hemianopia but in *crossed amblyopia*, a concentric diminution of fields of vision, greater on side opposite to lesion. *Mind-blindness* may occur, failure to recognize nature and use of objects. Pupil reflex normal.

Hemianopia occurs also in *migraine* and *hysteria*.

III. THE OCULOMOTOR NERVES (THIRD, FOURTH, AND SIXTH).

Anatomy.—

THIRD NERVE (*Oculomotor*).—*Origin*: Nuclei in floor of aqueduct of Sylvius. Emerges at inner side of crus, just in front of pons. *Distribution*: (1) Superior branch: levator palpebræ and superior rectus. (2) Inferior branch: internal and inferior recti and inferior oblique. (3) Constrictor of iris. (Each muscle has a separate nucleus, which may alone be affected by lesions.)

FOURTH NERVE (*Trochlear*).—*Origin* Nucleus in floor of aqueduct of Sylvius. *Distribution*: Supplies superior oblique muscle.

SIXTH NERVE (*Abducent*).—*Origin* Nucleus in floor of fourth ventricle. Emerges between pons and medulla. *Distribution*: Supplies external rectus muscle. Sends fibres to third nucleus (*see below*, CONJUGATE DEVIATION); also has connections with eighth nucleus, concerned in equilibrium.

General Symptoms of Paralysis of External Ocular Muscles.—

1. LIMITATION OF MOVEMENT.—In direction of paralyzed muscle. Later, affected and increased by contraction of unopposed antagonistic muscle.

2. STRABISMUS OR SQUINT.—Visual axes not in correspondence: (a) *Convergent*, when axes cross (e.g., paralysis of external rectus); (b) *Divergent*, when axes diverge (e.g., paralysis of internal rectus).

Primary deviation is deviation of axis of affected eye from parallelism with that of normal eye.

3. SECONDARY DEVIATION.—Method of demonstration: Patient looks at object in position involving use of affected muscle (e.g., right internal rectus). Sound (left) eye is now covered from sight of object, when it moves further outwards: due to the excessive nerve effort to contract the weak muscle affecting both the two muscles which act together (right internal and left external rectus). Absent in spasmodic strabismus (ordinary squint).

Paralysis of External Ocular Muscles, *continued.*

4. ERRONEOUS PROJECTION.—Example: Paralysis of right internal rectus; object placed to left of mid-position. On looking at object, nerve effort to move eye is greater than normal, and brain is deceived as to amount of movement; hence on attempting to touch object, finger passes to *left* of it.
   Giddiness (ocular vertigo) is often present, since maintenance of equilibrium partly depends on estimation of relations of surrounding objects.
5. DIPLOPIA.—True image seen by sound eye and false by affected eye: (*a*) *Simple* or *homonymous* diplopia: in convergent strabismus. False image on same side as the (affected) eye by which it is seen. (*b*) *Crossed* diplopia: in divergent strabismus.

Werner's 'Artificial Memory' assists identification of affected muscle. Place coloured glass before one eye to identify images during examination.

Lesions of the Motor Nerves of the Eyeball.—Symptoms vary: (1) According as the nerves are affected together, separately, or partially (especially the third); (2) With the site of affection. This site may be:—

a. NUCLEAR OR SUPRANUCLEAR, from nucleus to cortex. Usually more than one nucleus is involved. Affects movements rather than individual muscles, e.g., conjugate deviation (*see below*). Nuclear lesions usually arise from chronic degenerations; supranuclear lesions from hæmorrhage, etc.

b. NERVE BETWEEN NUCLEUS AND EMERGENCE AT BASE OF BRAIN, e.g., lesion of crus (third nerve), pons (sixth nerve). Other structures also affected. Cause: tumour, gumma; basal meningitis.

c. NERVE IN COURSE AT BASE OF BRAIN.—Cause: meningitis, gumma, aneurysm, tumour; also, in third nerve, neuritis from diphtheria or tabes. Nerves often affected separately or partially.

d. NERVE IN OR NEAR ORBIT.—Fractures, disease of bones, thrombosis of cavernous sinus, etc. May affect all nerves.

Total ophthalmoplegia results if all nerves are completely paralyzed.

THIRD NERVE.—Lesions cause either paralysis or spasm.

CAUSES.—(1) Meningitis, etc., at base, or in substance of brain, especially syphilis; (2) Diphtheria, tabes.

COMPLETE PARALYSIS.—Superior oblique and external rectus unaffected; hence eye can be moved out and slightly down and outwards. Other symptoms are: (1) Divergent strabismus; (2) Ptosis; (3) Diplopia; (4) Pupil dilated, no reaction to light, or power of accommodation.

PARTIAL PARALYSIS more common. Various types, especially *ptosis*, or internal ocular muscles only.

LESION OF ONE CRUS may produce: (1) Paralysis of face and limbs on opposite side; with (2) Paralysis of third nerve on same side, often partial, e.g., *ptosis*. (*See* p. 855.)

Recurrent Oculomotor Paralysis.—Rare condition. Attacks accompanied by headache and vomiting: related to migraine.

FOURTH NERVE.—Rarely affected alone.

PARALYSIS causes *diplopia* on looking downwards and inwards, also slightly deficient movement in same direction, but difficult to recognize; head is inclined downwards, with chin towards sound side.

SIXTH NERVE.—Injury at base of brain common, owing to long course.

PARALYSIS produces: (1) Movement outwards lost or impaired; (2) Convergent strabismus; (3) Diplopia towards affected side; (4) Head turned towards affected side.

'CONJUGATE DEVIATION'.—From the *nucleus* of the sixth nerve, fibres for the supply of the opposite internal rectus run to the nucleus of the opposite third nerve and then in its trunk. Hence: (1) In a nuclear lesion of the sixth nerve, both eyes deviate (conjugate deviation) to the opposite side, looking away from the lesion; (2) In a supranuclear lesion (*see* CEREBRAL HÆMORRHAGE), eyes look towards the lesion; (3) If lesions produce spasm (less common) and not paralysis, directions are reversed.

LESIONS OF THE PONS.—The sixth *nucleus* is in propinquity to the *fibres* of the seventh nerve. Hence a lesion of one side of the pons may produce: (1) Facial paralysis on the same side; with (2) Conjugate deviation to the opposite side. (*See* p. 856.)

Lesions of the Internal Ocular Muscles.—The muscles are: (1) *Ciliary muscle*, concerned in accommodation (supplied by third nerve); (2) *Constrictor of the iris* (third nerve); (3) Dilator of the iris (cervical sympathetic).

CYCLOPLEGIA.—Loss of power of accommodation from paralysis of ciliary muscle. Distant objects clear, near objects indistinct (corrected by convex lenses). Occurs in: (1) *Diphtheria*—common; (2) Tabes—rarely; (3) Degeneration of nucleus.

IRIDOPLEGIA.—Occurs in following forms:—

1. LOSS OF REFLEX TO LIGHT.—Due to interruption of arc (for path, *see* WERNICKE'S HEMIOPIC REACTION, p. 832). With presence of reaction to accommodation, constitutes *Argyll Robertson pupil* (tabes and, less commonly, dementia paralytica).

2. ACCOMMODATION IRIDOPLEGIA.—Usually with cycloplegia, e.g., in diphtheria.

3. LOSS OF SKIN REFLEX.—On pinching skin of neck, pupil normally dilates from stimulation of cervical sympathetic: absent occasionally in lesions of cervical sympathetic, cervical cord, or medulla.

Lesions of the Internal Ocular Muscles, *continued.*

4. PARALYSIS OF DILATOR OF IRIS.—Small pupils occur in spinal disease, e.g., spinal myosis in tabes. Unilaterally, in lesions of cervical sympathetic—e.g., in brachial plexus injuries—with narrowing of palpebral fissure.

Ophthalmoplegia Externa.—Paralysis of the external muscles of the eye: slow, chronic, bilateral, and may progress to completeness. *Cause*: A chronic degeneration of the nuclei; may be associated with bulbar paralysis or progressive muscular atrophy; rarely, tabes, tumours, basal meningitis (syphilitic). *Symptoms*: Usually commences with ptosis; finally no movements.

OTHER VARIETIES.—*Acute* forms rare: (1) Vascular lesions, often syphilitic; (2) Inflammation (polioencephalitis superior, often fatal). Rarely in diphtheria and other causes of multiple neuritis.

Ophthalmoplegia Interna.—Progressive paralysis of internal ocular muscles, usually with ophthalmoplegia externa; together form *total ophthalmoplegia.*

Ptosis.—Origin may be:—

1. Congenital.
2. Lesions of the third nerve. Often with affection of other ocular muscles, and *frequently due to syphilis.* Also in cerebral lesions.
3. Hysteria: bilateral ptosis.
4. Lesions of cervical sympathetic nerve. Ascribed to paralysis of unstriped muscle in upper lid. Pupil is contracted. (Pseudo-ptosis.)
5. Myasthenia gravis.
6. 'Matutinal ptosis': for few hours after waking, in delicate women.
7. Muscular wasting of facial muscles.
8. Pain, as in migraine (transient).

Nystagmus.—Rapid involuntary bilateral rhythmical oscillations of the eyes. Direction of movement lateral, rarely rotary or vertical. Usually absent in mid-position, occurring when eyes are moved laterally. Particularly connected with disease in mid-brain and cerebellum. Common conditions:—

1. Tumours of cerebellum, pons, and corpora quadrigemina (not of cerebral cortex),
2. Disseminated sclerosis.
3. Friedreich's ataxia.
4. Disturbances of semicircular canals—e.g., Ménière's disease.
5. 'Head-nodding' in children: occurs in mid-position of eyes.
6. Albinos.
7. Coal-miners.
8. Opacities of cornea, errors of refraction: occasionally.

Pupils: Abnormalities of Size and Shape.—

SIZE OF PUPIL depends upon: (1) Oculomotor nerve (constrictor of iris muscle); (2) Cervical sympathetic nerve (dilator

of iris muscle); (3) Spiral arteries of iris—as these straighten under high pressure, the pupil diminishes, and vice versa. *Dilatation* results from either (*a*) paralysis of third nerve, (*b*) irritation of cervical sympathetic, or (*c*) low blood-pressure. Conversely, for *contraction*, (*a*) irritation of third nerve, (*b*) paralysis of cervical sympathetic, and (*c*) high blood-pressure.

DILATED PUPILS (especially with low blood-pressure).—Causes:—

1. Anæmia.
2. Aortic regurgitation.
3. Increased cerebral pressure: tumours, hæmorrhage, abscess, meningitis.
4. Drugs: atropine, cocaine, alcohol. Also daturine, duboisine.
5. Emotion.
6. Asphyxia.
7. Stimulation of cervical sympathetic nerve.

CONTRACTED PUPILS (especially in high blood-pressure).—Causes:—

1. Chronic interstitial nephritis and arteriosclerosis.
2. Irritation of third nerve nucleus: hæmorrhage in pons.
3. Spinal myosis—e.g., in tabes.
4. Drugs: morphia, eserine, pilocarpine.
5. Venous congestion e.g., in bronchitis, whooping-cough.

Also *occur physiologically* in bright light, in accommodation, and during sleep.

IRREGULAR PUPILS.—Sequel to iritis.

UNEQUAL PUPILS.—Causes:—

1. Dementia paralytica.
2. Third-nerve lesion, paralysis or irritation: gummatous meningitis.
3. Thoracic aneurysm (q.v.).
4. Cervical sympathetic lesion, paralysis or irritation: trauma to brachial plexus; rarely tumours in neck, pleural diseases.

Also: glass eye, atropine in one eye.

IV. FIFTH NERVE.

(*Nervus Trigeminus.*)

Lesions.—

1. IN PONS.—From hæmorrhage or softening, tumours; very rarely, bulbar paralysis, disseminated sclerosis.
2. AT BASE OF BRAIN.—From meningitis, tumours, caries of petrous bone. *In fractures usually escapes.*
3. DISTAL TO GASSERIAN GANGLION.—First division. From tumours affecting cavernous sinus; aneurysm of internal carotid; cellulitis, etc., of orbit. Second and third divisions: growths of sphenomaxillary fossa.

SUPRANUCLEAR LESIONS occur rarely in lesions of posterior portion of internal capsule, and hence with anæsthesia of limbs; motor portion of nerve escapes.

Lesions of the Fifth Nerve, *continued*

Symptoms.—

SENSORY PORTION.—

1. *Loss of sensation* half of face and scalp, of anterior two-thirds of tongue (to circumvallate papillæ), of soft and hard palate and upper lip, and of nose; also conjunctiva. Epicritic slightly less than protopathic loss. (On drinking, cup feels broken.)
2. *Tingling* and pain: may precede loss of sensation.
3. *Secretions diminished*—buccal, nasal, and lachrymal.
4. Sense of smell affected, from absence of secretion.

Movements of face muscles awkward, owing to loss of deep sensibility.

Ulceration of cornea frequent, from injury when insensitive.

Herpes zoster in area of ophthalmic branch may occur, and subsequent neuralgia.

MOTOR PORTION (in infranuclear lesions only).—

1. Loss of power in muscles of mastication on affected side, viz., temporal, masseter.
2. Inability to move jaw towards sound side; on depression, jaw deviates to affected side (external pterygoid muscle).

Mastication is possible by muscles of sound side.

Spasm of muscles (*see* FACIAL SPASM, p. 913).

Sense of Taste: Note on Path of Impulses.—Loss of sense of taste over anterior two-thirds of tongue possibly follows paralysis of fifth nerve. The course of fibres transmitting gustatory sensations to the brain is in dispute.

1. ANTERIOR TWO-THIRDS OF TONGUE.—By lingual nerve, chorda tympani, and facial nerve, to geniculate ganglion. Further course, possibly. (a) Great superficial petrosal nerve to Meckel's ganglion and the second division of the fifth nerve (probable course); or (b) By the pars intermedia with the facial nerve.
2. POSTERIOR THIRD OF TONGUE.—Either (a) By the glossopharyngeal nerve to the petrous ganglion, thence by Jacobson's nerve and the small superficial to the otic ganglion and the third division of the fifth nerve (hence it is possible that all taste fibres end in the fifth nucleus); or (b) Direct in the glossopharyngeal nerve.

Possibly these paths are alternatives.

TRIGEMINAL NEURALGIA.

(*Tic Douloureux. Epileptiform Neuralgia. Neuralgia Major.*)

Paroxysmal pain in the course of branches of the fifth cranial nerve in the absence of recognizable lesions.

Etiology.—

AGE AT ONSET.—Middle life.

SEXES.—About equal.

EXCITING CAUSE OF ATTACKS.—Of first attack, none. Subsequently: *change of weather*, constipation, worry, debility, or none.
NO NEUROTIC FACTOR.

Morbid Anatomy.—Origin is in, or distal to, Gasserian ganglion, but histology normal, or slight fibrosis. *Pathogenesis* unknown.

General Characteristics.—

1. DISTRIBUTION OF PAIN.—Usually second or third branches; ophthalmic branch rare and late. Pain in course and area of nerve: may be superficial tenderness. Never commences outside area of nerve, but during attack may spread to neck, behind ear, and occipital region. *Very rarely bilateral.*
   OPHTHALMIC BRANCH.—Rarely affected. Pain from supra-orbital notch over scalp.
   SECOND BRANCH (*infra-orbital neuralgia*).—Pain between orbit and mouth. Tender spots at side of ala nasi, infra-orbital foramen, along gums, malar bone.
   THIRD BRANCH.—Pain in lower jaw, often tongue, and later ear and temple. Tender spots at mental foramen and in front of ear.
2. COMMENCES "just under the skin", and radiates thence through course of nerve.
3. COMMONEST POINTS OF ORIGIN.—(*a*) Just external to ala nasi; (*b*) Infra-orbital foramen; (*c*) Mental foramen (below canine tooth).
4. CHARACTER OF PAIN.—Paroxysmal; in later stages agonizing, resembling "red-hot needles".
5. DURING ATTACK, repeated paroxysms occur: of duration few seconds to minutes. Often numerous, day and night. Follow trivial stimuli, e.g., eating, speaking, draughts.
6. ATTACKS RECUR, with remissions. Intervals diminish; intensity increases; distribution spreads.
7. NO TENDENCY TO CESSATION OF ATTACKS.

VARIOUS SYMPTOMS, vasomotor, trophic, often occur during paroxysm: (1) Local flushing and sweating; (2) Lachrymation, salivation, thin nasal discharge; (3) Twitching of facial muscles (face also often drawn up from pain). After repeated attacks: skin becomes shiny, hair in area may be gray or rubbed away. Mouth often foul (from fear of cleansing), and teeth removed.

Treatment.—

IN EARLY STAGES while diagnosis doubtful, try methods for minor neuralgia, especially gelsemium and butyl-chloral hydrate. Finally these become ineffective.

SPECIAL METHODS WHEN DIAGNOSIS IS FULLY ESTABLISHED.—(1) *Schlösser's treatment*: Injection of alcohol into nerve trunks on emerging from the foramina. Anæsthesia results in course of branch. Freedom from pain often for many months: can be repeated. May be tried before next method. (2) *Extirpation of Gasserian ganglion*: Results extremely good, and mortality becoming very low.

NEURALGIA MINOR OF THE FIFTH NERVE, AND ALLIED CONDITIONS.

A group varying from conditions resembling tic douloureux to simple 'headaches'. These must be excluded before diagnosing tic douloureux.

1. ORGANIC DISEASE AFFECTING FIFTH NERVE (*see* FIFTH NERVE). Symptoms may resemble tic douloureux.
2. NEURALGIA MINOR.—
   - *a.* Pain shooting along nerve: superficial tenderness, slight and only in distribution of nerve. From teeth, errors of refraction, iritis, etc.
   - *b. Referred pain*, characterized by *pain and superficial tenderness* in areas not corresponding to peripheral nerves. Due to teeth and other local causes.
   - *c.* Secondary to disease elsewhere in body.
   - *d.* In general debility, anæmia, neurasthenia, etc.

V. SEVENTH OR FACIAL NERVE.

Anatomy.—*Nucleus* in pons, in floor of fourth ventricle. *The fibres* wind round nucleus of sixth nerve, then lie close to fibres from the cerebral cortex on their way to the decussation in the medulla, and emerge between pons and cerebellum. The seventh and eighth nerves pass to the internal auditory meatus; the seventh enters the aqueduct of Fallopius, and, after its intrapetrous course, emerges at the stylomastoid foramen.

Paralysis of the Facial Nerve.—The facial nerve may be affected at numerous sites; lesions of other structures aid in localization.

1. SUPRANUCLEAR LESIONS.—In cortex, corona radiata, internal capsule, or rarely upper portion of pons. *Causes*: Tumours, abscess, hæmorrhage, or softening. *Special characters*: (i) Upper branch unaffected—viz., frontalis, orbicularis palpebrarum, corrugator (supplied by fibres from third nucleus); (ii) Hemiplegia usually present; (iii) Paralysis of upper motor neuron type. If 'conjugate deviation' present, looks *towards* the side of the lesion (except in early spasm—*see* p. 855). Voluntary movements affected more than emotional.
2. NUCLEAR LESIONS.—Generally as part of bulbar paralysis, and other nuclei are affected: upper fibres generally escape, otherwise of lower motor neuron type. Occasionally in diphtheria. Also in lesions of pons.
3. INFRANUCLEAR LESIONS.—Entire peripheral nerve affected; lower motor neuron type.
   - *a.* IN PONS.—*Special characters*: (i) '*Crossed paralysis*' common—face on side of lesion, arm and leg on opposite side; (ii) Nucleus of sixth nerve almost always affected—'*conjugate deviation' away from lesion.* Often fifth nerve also involved.

b. At Base of Brain.—*Causes*: Cerebellopontine tumours, gumma, meningitis, occasionally fractures. *Special characters*: Eighth nerve usually affected (note that deafness with facial paralysis also occurs from otitis media).

c. Within Temporal Bone.—*Causes*: Especially caries in otitis media, occasionally operations on mastoid. *Special characters*: Taste lost in anterior two-thirds of tongue, unless lesion below separation of chorda tympani (rare); if stapedius affected, hypersensitiveness to musical tones.

d. Peripheral Nerve (Bell's palsy).—Common. Causes:—

(i) Injuries and blows close to foramen, operations on tumours, forceps at birth.

(ii) 'Cold' or 'rheumatic' form; commonest variety; constitutes true 'Bell's palsy'. Is a parenchymatous neuritis, probably swelling and compression of nerve within Fallopian aqueduct.

(iii) Syphilis: not uncommon cause.

Tetanus is occasional cause; *mumps never*.

Facial diplegia rare. From double otitis media, lesions of pons or base of brain, rarely diphtheria.

Symptoms of Paralysis of Peripheral Nerve, or 'Bell's Palsy'.—(Characteristics of lesions at special sites are referred to above; note especially supranuclear lesions.)

ONSET.—In 'rheumatic' form, sudden; maximum paralysis within twenty-four hours. In otitis media, onset more gradual.

Loss of power on affected side; both voluntary and emotional movements. *Skin* smooth, may be slight swelling. *Pain* near ear occasionally. *Paralysis* of lower motor neuron type.

CHARACTERISTICS.—

(1) Eye cannot be closed (orbicularis palpebrarum). In attempting closure, eyeball turns up and outwards (inferior oblique).

(2) Forehead cannot be wrinkled (frontalis). Frowning lost (corrugator).

(3) In 'showing the teeth', lips not separated on affected side.

(4) In smiling, affected side unresponsive.

(5) Whistling impossible.

(6) On protruding tongue, lips drawn to sound side, hence tongue appears to be towards paralyzed side, but is median to teeth.

OTHER FEATURES.—Food collects in cheek (buccinator). Fluid runs out while drinking. Nostril falls in during inspiration. Conjunctiva liable to injury, lower lid droops, epiphora common. Speech slightly affected. Reflexes absent. If stapedius paralyzed, oversensitiveness to musical tones.

SENSATION UNAFFECTED, except in small area near and including external auditory meatus.

(*Note.*—This area of anæsthesia proves that the seventh is a mixed nerve, but exact sensory functions still in dispute.)

Taste lost in anterior two-thirds of tongue, if chorda tympani involved. (For *paths of taste*, *see* Fifth Nerve.)

Bell's Palsy, *continued*

IN OLD-STANDING CASES.—Wrinkles more marked on affected side from muscular contractures, and until tests are performed the sound side appears to be paralyzed. Reaction of degeneration present.

Course and Prognosis.—When due to 'cold', recovery usual and may be complete. From injury, paralysis more often permanent. With other factors, varies with cause.

ELECTRICAL REACTIONS.—(1) If no change within two weeks, recovery usual in three to four weeks; (2) If reaction of degeneration present after three months, recovery rarely complete. Various intermediate changes and prognoses.

RECOVERY may commence up to three months after lesion, and, once commenced, improvement may continue for twelve to eighteen months.

Treatment (of Bell's palsy from 'cold').—

AT ONSET.—Hot fomentations over ear, or small blister *over mastoid*. Free purge. Sodium salicylate and potassium iodide.

AFTER ONE TO TWO WEEKS, commence: (1) *Galvanic current*, quarter-hour, daily; positive pole behind ear, negative pole stroked over muscles. (2) Massage.

CAUSAL CONDITIONS must be treated. If syphilis, usual methods. If nerve divided at operation, ends to be united if possible: immediately, if discovered at time; otherwise may wait until wound healed. Otitis media: indicates complete mastoid operation.

NERVE ANASTOMOSIS.—Indications justifying this operation are: (1) Improvement not commencing six months after lesion; (2) Severity of affection. Anastomosis preferably with hypoglossal nerve; at first tongue contracts with face, but dissociated movements become established. Spinal accessory nerve less advisable; causes twitching of shoulder.

Spasm of Facial Muscles.—*See* FACIAL SPASM, p. 913.

VI. EIGHTH OR AUDITORY NERVE.

The auditory nerve consists of two separate parts: (1) The *cochlear* nerve, concerned in hearing; (2) The *vestibular* nerve, concerned with equilibrium.

1. COCHLEAR NERVE.

Anatomy.—From the organ of Corti in the labyrinth and the spiral ganglion, fibres run to the two nuclei of the cochlear nerve in the floor of the 4th ventricle; whence: (1) From the tuberculum acusticum, fibres pass anterior to the restiform body in the striæ acusticæ, and after decussation reach the lateral fillet; (2) From the ventral nucleus, fibres pass posterior to the restiform body, through the superior olive to the lateral fillet. From the latter, paths lead to the posterior corpus quadrigeminum and to the median geniculate body, and thence through the internal capsule

to the cerebral cortex (temporal gyrus). Through the superior olive are communications with the third, fourth, and sixth nuclei, connecting hearing with eye movements.

Site of Lesions.—

LESIONS OF CENTRE IN CORTEX, superior temporal gyrus, produce, not deafness, but (if on left) word-deafness—viz., meaning of words not understood.

LESIONS OF NERVE AT BASE OF BRAIN arise from: tumours, especially cerebello-pontine; fractures; hæmorrhage, meningitis, especially cerebrospinal. Tabes may affect the nuclei.

Symptoms Resulting from Lesions.—(1) Hyperæsthesia or altered function; (2) Deafness or diminished function.

1. HYPERÆSTHESIA OR HYPERACUSIS.—Sounds heard with unusual intensity. Rare. In hysteria, and rarely in cerebral lesions. With paralysis of stapedius (seventh nerve), low musical tones are very intense.

DYSACUSIS.—Intolerance of sounds; occurs in headache, neurasthenia, and in some cerebral lesions.

TINNITUS AURIUM.—Subjective sensations of ringing, etc., in the ear.

Causes include: (a) Wax on drum; (b) Anæmia; (c) Neurasthenia; (d) Certain drugs, e.g., quinine, salicylates; (e) Middle-ear disorders; (f) Epileptic aura (rare); (g) Exposure to loud noises, and (h) Labyrinthine disturbances, as in Ménière's disease. Possibly also gout, migraine.

Varieties.—May be (a) Continuous; (b) Pulsating tinnitus (rarely due to aneurysm); less commonly (c) 'Clicking', probably clonic spasm of tensor tympani.

Treatment.—Examine ear for local causes. Treat any general factors. Tonics or potassium bromide to be tried.

2. DEAFNESS OR DIMINISHED FUNCTION.—*Causes*: (a) Outer ear—wax on drum; (b) Middle-ear conditions; (c) Internal ear, affecting labyrinth. The last may be primary, or secondary from middle ear, and includes: (i) Inflammations; (ii) Scleroses from syphilis, mumps, and rarely other infectious diseases; (iii) Cerebrospinal meningitis, tumours and fractures affecting nerve; (iv) Gout, diabetes, nephritis; (v) Hæmorrhage and effusions; (vi) From quinine and salicylates, transient.

TUNING-FORK TESTS FOR NERVE DEAFNESS.—

1. *Weber's Test.*—Place on forehead; loudest on deaf side if conducting apparatus affected, but loudest on sound side if nerve affected.
2. *Rinné's Test.*—Tuning-fork vibrating placed on mastoid; when no longer audible is held to external auditory meatus; if audible, proves 'nerve-deafness' ('positive Rinné test').

In 'nerve-deafness', hearing better in quiet place. In deafness from middle-ear disease, hearing better amid noise

2. VESTIBULAR NERVE.

Anatomy.—From the lining membrane of the semicircular canals, filaments run to the ganglion vestibulare, and thence to the brain. Here they enter: (1) *Deiters' nucleus* (lateral eighth nucleus); thence fibres run in the middle peduncle to the 'roof nuclei' of the cerebellum, here connecting with motor tracts to the muscles. (2) *Median nucleus*; the fibres from here decussate; some (*a*) enter posterior longitudinal bundles connecting with third, fourth, sixth nuclei; others (*b*) enter tegmentum and pass through internal capsule to cerebral cortex. (3) The nucleus of the descending root; to vestibulo-spinal tract. The vestibular nerve is thus connected with important structures controlling equilibrium; it also receives fibres from the labyrinth.

Symptoms Resulting from Lesions.—*Vertigo especially*; also loss of co-ordination of muscles of head, neck, and eyes; tinnitus and deafness are not uncommonly present (cochlear nerve); occasionally nystagmus.

Vertigo.

Among numerous causes are: (1) Rapid changes of position, especially rotary; (2) Disturbances of alimentary canal, heart, kidney, circulation, etc., including high and low blood-pressure; (3) Ocular defects; (4) Alcohol, tobacco in excess, and other drugs; (5) Diseases of the brain, markedly in cerebellar lesions; (6) Epilepsy; (7) Migraine; (8) Numerous ear conditions (many of the previous causes are also directly due to action on labyrinthine pressure).

Auditory Vertigo.—Vertigo results from any condition affecting the pressure of the endolymph in semicircular canals, and thus disturbing the mechanism of equilibrium, or causing irritation of vestibular nerve. Causes resemble those of deafness (q.v.).

Labyrinthitis.

Actual disease of the labyrinth occurs in two forms: (1) True *Ménière's disease, acute primary labyrinthitis*; (2) *Ménière's symptom-complex*, labyrinthitis chronic and secondary, e.g., to middle-ear disease.

1. MÉNIÈRE'S DISEASE.—Acute labyrinthitis.
   - CAUSE.—Hæmorrhages, effusions, or acute inflammation o labyrinth.
   - AGE.—Past middle age.
   - ONSET.—Sudden.
   - SYMPTOMS.—(*a*) *Vertigo*: patient falls to ground; may be transient unconsciousness. (*b*) *Tinnitus*: onset may precede vertigo. (*c*) *Nausea*, pallor, cold sweat, and vomiting follow; may be collapse. (*d*) *Deafness* then noted. Nystagmoid movements may occur, away from affected side. Paroxysms recur at irregular intervals. *Deafness progressive.* Tinnitus may persist, and *psychical changes* develop.
2. MÉNIÈRE'S SYMPTOM-COMPLEX.—
   - CHARACTERISTICS.—(*a*) *Tinnitus*; (*b*) *Attacks of vertigo*; with (*c*) *Nausea*; (*d*) *Progressive deafness.*

DIAGNOSIS OF MÉNIÈRE'S SYNDROME.—From: (*a*) *Epilepsy* by tinnitus and progressive deafness and absence of micturition or biting of tongue. (*b*) Gastric, oculai, or cardiac vertigo: rarely so severe; no progressive deafness (may be vomiting). (3) Intracranial tumours, especially cerebellar: difficult. Barany's tests of great assistance.

TREATMENT.—Unsatisfactory. Bromides or iodides of most effect. Amyl nitrite in high blood-pressure. Operations on semicircular canals as final resort.

VII. NINTH OR GLOSSOPHARYNGEAL NERVE.

Probably a portion of the vagus. Distribution: (1) *Motor*: Stylo-pharyngeus and middle constrictor of pharynx. (2) *Sensory*: Upper pharynx. (3) *Taste* (*see* FIFTH NERVE). Little known of isolated lesions.

VIII. TENTH, VAGUS, OR PNEUMOGASTRIC NERVE.

Site and Cause of Lesions.—

1. NUCLEUS.—Bulbar paralysis, tabes (crises). Rarely syringomyelia, disseminated sclerosis
2. AT BASE OF BRAIN.—Tumours, meningitis (especially syphilitic), aneurysm.
3. IN NECK.—Operations, wounds, tumours
4. IN THORAX.—Especially in thoracic aneurysm, usually left recurrent laryngeal nerve, on right rarely in pleural adhesions.
5. NEURITIS.—Diphtheria, rarely in alcohol, influenza, arsenic.

Distribution of Nerve.—Very extensive pharynx, larynx, heart, lungs, stomach.

Total Unilateral Paralysis.—Principal results. (1) *Paralysis of palate*; shown on movement only; may be partial anæsthesia of palate and pharynx. (2) *Paralysis of pharyngeal muscles*; symptoms slight. (3) *Vocal cords* in 'cadaveric position', voice nasal or hoarse, cough weak and harsh

Pharyngeal Branches.—

BILATERAL PARALYSIS, usually from bulbar paralysis or diphtheria. Difficulty in swallowing, food may enter larynx or nares (if palate paralysed).

SPASM OF PHARYNX.—In hydrophobia. Functional in hysteria and pseudo-hydrophobia.

Laryngeal Branches (*see also* THORACIC ANEURYSM, p 719).—

PARALYSIS usually partial: abductors affected before adductors. Larynx must be examined when involvement possible, as symptoms are absent in early stages.

1. BILATERAL ABDUCTOR PARALYSIS (posterior crico-arytenoid muscles).

   Cause.—(*a*) Lesions in nuclei—bulbar paralysis, tabes. (*b*) Pressure on both vagi or recurrent laryngeals. Rarely in laryngeal catarrh, hysteria.

Paralysis of Tenth, Vagus, or Pneumogastric Nerve, *continued.*

Symptoms.—(a) *Inspiratory stridor,* expiration normal; (b) *Voice unaffected,* coughing normal; (c) *Dyspnœa*—often dangerous, tracheotomy may be necessary.

On Examination of Larynx.—Vocal cords almost in position of phonation, no movement on inspiration. Adductors involved later.

2. UNILATERAL ABDUCTOR PARALYSIS.—

Cause.—Usually from thoracic aneurysm.

Symptoms.—Voice generally hoarse or altered; no dyspnœa.

On Examination.—Vocal cord on affected side shows no movement on inspiration.

3. ADDUCTOR PARALYSIS (lateral crico-arytenoid and arytenoid muscles).—

Cause.—Hysteria.

Symptoms.—Aphonia, no stridor or dyspnœa.

On Examination.—Cords do not approximate on phonation.

SPASM OF MUSCLES OF LARYNX.—In children, adductor spasm occurs in laryngismus stridulus. In adults, in rare laryngeal crises of tabes, and rarely in hysteria.

ANÆSTHESIA OF LARYNX.—In bulbar paralysis, diphtheria. Food may enter larynx.

Lesions of Branches to the Heart and Viscera.—Do not produce characteristic symptoms.

CARDIAC BRANCHES.—Vagus is cardio-inhibitory. Irritation slows the heart rhythm, paralysis accelerates it.

GASTRIC AND ŒSOPHAGEAL BRANCHES.—Muscular movements controlled by vagus—vomiting, etc. Gastric crises in tabes due to irritation of nuclei.

IX. ELEVENTH OR SPINAL ACCESSORY NERVE.

Anatomy.—Consists of two portions: (1) *Accessory portion*: Nuclei in medulla, continuous with vagal nuclei. Rejoins vagus and supplies muscles of larynx and pharynx. (2) *Spinal portion*: From anterior horns of first to fifth cervical segments. Fibres pass up through foramen magnum. Supplies sternomastoid and trapezius muscles. Nerve leaves skull through jugular foramen.

Site and Cause of Lesions.—

NUCLEI.—In bulbar paralysis. Spinal portion in progressive muscular atrophy. Occasionally in syringomyelia.

AT BASE OF BRAIN.—Caries of vertebræ, tumours, meningitis.

PERIPHERAL NERVE.—Wounds, cervical caries, etc.

Paralysis of Sternomastoid (unilateral).—Rotation of head to other side impaired. No deformity. Muscle wastes.

Paralysis of Trapezius.—Upper portion suffers most: deficient movement of scapula on deep breath or shrugging shoulder. If middle portion weakened: shoulder drops, power of lifting arm

impaired. Paresis of lower portion resembles serratus magnus paralysis: angle of scapula rotated inwards by rhomboids and levator anguli scapulæ. (Trapezius is also supplied by third and fourth cervical nerves.)

Bilateral Paralysis.—If trapezii affected, head falls forward, as in progressive muscular atrophy. If sternomastoids affected, head falls back.

Spasm of Sternomastoid and Trapezius (*Torticollis*).—*See* SPASMODIC TORTICOLLIS, p. 915

X. TWELFTH OR HYPOGLOSSAL NERVE.

Motor nerve of the tongue. Nucleus in medulla, in floor of fourth ventricle.

Site and Cause of Lesions.—

1. SUPRANUCLEAR AND CORTICAL.—Common in *hemiplegia.*
2. NUCLEAR.—*Bulbar paralysis*, tabes; rarely in syringomyelia, disseminated sclerosis. Usually bilateral.
3. INFRANUCLEAR. Tumours, meningitis, fractures, callus.

Symptoms.—

1. UNILATERAL NUCLEAR OR INFRANUCLEAR PARALYSIS.—(*a*) *Paralysis of tongue*: On protrusion, drawn by sound geniohyoglossus towards affected side. (*b*) *Atrophy of tongue*: Unilateral. May be reaction of degeneration. (*c*) *Mucous membrane of tongue in folds.*
2. NUCLEAR LESIONS.—Usually bilateral: tongue immobile, speech and mastication difficult. Orbicularis oris, supplied by fibres from twelfth running in seventh nerve, usually paralyzed in nuclear lesions.
3. SUPRANUCLEAR LESIONS.—Wasting slight, hemiplegia rarely absent.
4. LESIONS IN MEDULLA.—Pyramidal tract usually involved, whence 'crossed paralysis', viz., limbs on one side, and tongue on other; on protrusion, tongue deviates towards *sound limbs.*

CHAPTER CXXX.

DISEASES OF THE CEREBRUM.

I. APHASIA.

Disorders of speech result from *lesions of special speech centres* in the cerebral cortex and of association fibres deep to those centres, and also from lesions of the *motor cortical centres* and paths connecting them with the muscles of articulation.

Note on Theories of Aphasia.—Speech is necessarily dependent on many factors, anatomical, physiological, and intellectual. Numerous theories have been evolved from different aspects and have produced prolonged controversies. The views here

Aphasia, *continued.*

followed are those of the 'diagrammatists'. Over many years they have answered the purposes and tests of the clinician by diagnosing correctly the site of a lesion. But many cases fail to 'fit in' with the hard-and-fast centres, and the theory is manifestly imperfect, taking no note of well-known factors of intellect and emotion. Marie, in 1905, denied the existence of all centres, including Broca's area, and considered aphasia to be invariably an impairment of intellect due to lesions of a region specialized for language in general, the 'zone of Wernicke' in the angular and supramarginal gyrus and posterior ends of the first and second temporal convolutions. Head more recently has studied aphasia from the aspect of disturbance of intellect, and regards it as an interference with expression of thought, speech being regarded as an entity, and indivisible into the watertight compartments of motor, sensory, and other diagrammatic aphasias. As there is obviously truth in all these opposing views, no satisfactory explanation of aphasia can be expected until they have been fitted together.

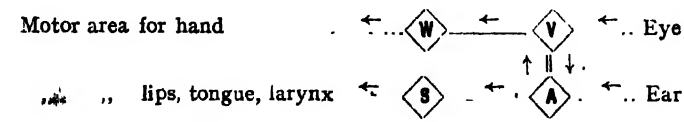
Speech Centres.—Four special centres (arranged in the order of development in a child, and in sites for right-handed subjects):—

1. AUDITORY SPEECH CENTRE (A).—In first left temporal convolution. Centre for memory of sounds of words, i.e., ability to understand meaning of words heard.
2. MOTOR SPEECH CENTRE, OR GLOSSO-KINÆSTHETIC CENTRE (S).—In 'Broca's area', third left frontal convolution, and thus close to motor cortical centres for lips and tongue. Centre for production of speech.
3. VISUAL SPEECH CENTRE (V).—In angular and supramarginal gyri. Centre for meaning of written words, i.e., reading.
4. WRITING CENTRE, OR CHEIRO-KINÆSTHETIC CENTRE (W).—In second left frontal convolution, and thus close to motor cortical centre for hand.

(The centres may be briefly referred to by the letters placed against them.)

These centres fall into two groups: (i) *Motor speech centres*, S and W, concerned in performance of speech, spoken or written; (ii) *Sensory speech centres*, A and V, concerned in the reception and memory of speech. The former are close to the motor area, and hemiplegia is common in their lesions. The latter are close to the optic radiations, and sight may be affected.

The centres and their usual connecting tracts may be represented thus:—



'AUDITIVES' AND 'VISUALS'.—In most persons the memory of words depends mainly on auditory afferent impulses and is stored

in the auditory speech centre. In the rarer 'visual', the memory depends mainly on visual afferent impulses and is stored in the visual speech centre.

Types of Aphasia.—Depending on site of lesion, aphasia may be:
1. MOTOR APHASIA.—Lesions of speech and writing centres.
2. SENSORY APHASIA.—Lesions of auditory and visual centres: word-deafness and word-blindness.
3. ANARTHRIA, APHEMIA, ETC.—Lesions of the motor tract to the muscles concerned in articulation.

1. MOTOR APHASIA.

Characterized by *loss of voluntary speech.* Lesion in Broca's area

Complete Motor Aphasia.—
VOLUNTARY SPEECH.—Lost.
UNDERSTANDING OF SPEECH.—Retained, but some deficiency invariable.
AGRAPHIA.—Usually present: from proximity of writing centre (W).
'RECURRING UTTERANCES'.—Though speechless, is not wordless (Hughlings Jackson); may repeat a few inappropriate sentences; ascribed to centre in opposite hemisphere.
ALEXIA (loss of reading).—Usual in some degree.
All degrees occur to slight deficiencies, patient making mistakes in speech and recognizing the errors.

Agraphia.—Inability to write occurs in all grades of severity. Theoretically from lesion of cheiro-kinæsthetic area, but lesion rarely (if ever) localized, and *paralysis of hand* also exists from lesion of motor cortex. May be able to write from dictation.

Hemiplegia.—Usually present in motor aphasia, lesion including Rolandic area.

2. SENSORY APHASIA.

Defects in spoken or written speech due to lesions of the auditory and visual centres—causing defects in auditory or visual word-memory—or to lesions which destroy the afferent paths of auditory or visual speech stimuli. Lesions, then, may be: (1) In the centres (A or V); (2) Subcortical, involving the afferent paths to these centres, and the paths connecting the two hemispheres. The common defects are *word-deafness* and *word-blindness.*

Word-deafness or Auditory Aphasia.—Lesion of auditory speech centre may be complete or partial.
COMPLETE LESION OF CENTRE.—Disturbance of all forms of speech.
SYMPTOMS.—
1. Complete word-deafness: sounds convey no meaning.
2. Speech is a mere jargon of words. Reading and writing also lost.
3. Intellect disturbed.

In rare marked 'visuals', some voluntary speech may be

Sensory Aphasia, *continued.*

performed by a direct path V→S, the visual centre retaining the speech memories, and reading and writing may be to some extent retained. The prognosis in 'visuals' is thus better than in 'auditives'.

PARTIAL LESIONS OF CENTRE.—More frequent. All grades of severity occur, e.g. :—

1. Auditory word-memories can be revived by stimuli arriving at centre; thus patient *understands speech* and can read, while *voluntary speech is very slight*.
2. *Paraphasia*. In slighter degrees, voluntary speech present, but uses wrong words and is unaware of error. Understands, reads, and writes at dictation.

Amnesia verbalis (Bastian) is forgetfulness of words. An object may be described, e.g., 'something to write with' for 'pen'.

PURE WORD-DEAFNESS OR AUDITORY APHASIA.—*A subcortical lesion* may leave centre intact but isolated from afferent impulses. Extremely rare.

SYMPTOMS.—

1. Centre and efferent paths intact. Therefore: (i) Talks correctly (A intact); (ii) Reads aloud and can understand writing (V→A→S intact).
2. Afferent paths interrupted. Therefore (i) Does not understand speech; (ii) Cannot repeat words or write from dictation.

Word-blindness or Visual Aphasia.—Lesion in angular gyrus May affect centre or subcortical paths.

LESION OF VISUAL CENTRE.—

SYMPTOMS —

1. Unable to read; may recognize familiar portraits.
2. Understands speech.
3. Voluntary speech little affected.
4. Agraphia present, and unable to copy. Writing sometimes present in educated strong 'auditives', by direct A→W path, usually with 'paragraphia', i.e., writes wrong words; cannot read what he has written.

PURE WORD-BLINDNESS OR VISUAL APHASIA.—*A subcortical lesion* may leave centre intact but isolated from afferent impulses. Very rare.

SYMPTOMS.—

1. Centre and efferent paths intact. Therefore: (i) Understands speech; (ii) Voluntary speech normal; (iii) Writes, *but is unable to read* what is written.
2. Afferent paths interrupted. Therefore unable to read.
3. Right homonymous hemianopia (or rarely hemichromatopia) present from injury to optic radiations.

Word-deafness and Word-blindness.—May be combined in lesions affecting both centres. If complete, unable to understand, read, write, or speak. More commonly partial, with some degree of voluntary speech and communication by signs.

3. ANARTHRIA.

Disorders of articulation resulting from lesions of the paths conducting impulses from the motor centres to the muscles of tongue, lips, and larynx.

Lesions may occur at various sites :—

1. **Supranuclear Lesions.**—

BILATERAL LESIONS OF INTERNAL CAPSULE.—'*Pseudo-bulbar paralysis*'. Associated with double hemiplegia. The two lesions are usually not simultaneous. Affection of articulation permanent and may be complete.

BILATERAL LESIONS OF MOTOR CORTEX.—Less commonly.

SINGLE LESION OF MID-BRAIN AFFECTING BOTH TRACTS.—Rarely.

All these are lesions of upper motor neuron type : tongue firm and not wasted.

Transient and incomplete forms occur in unilateral lesions.

2. **Lesions of Nuclei and Lower Motor Neuron.**- Especially *bulbar paralysis*. Tongue wasted.

Disturbances of Co-ordination or ataxia of muscles of articulation may occur, e.g., in disseminated sclerosis, Friedreich's ataxia.

INVESTIGATION OF APHASIA.

Note.—In hemiplegia, hemianopia, etc., ascertain if patient is right-handed.

Series of Questions to Ascertain Lesion (based on plan of Beevor).—

1. Can he speak voluntarily and intelligently ? (S). Motor aphasia.
2. Can he understand what is said ? (A). Word-deafness.
3. Can he understand writing ? (V). Word-blindness.
4. Can he write spontaneously ? (W). Test for agraphia often difficult owing to paralysis of hand.
5. Can he repeat words ? (A → S).
6. Can he copy from print ? (V → W).
7. Can he pick out objects named ? (A → V).
8. Can he write from dictation ? (A → V → W).
9. Can he name objects seen, and read aloud ? (V → A → S).

PROGNOSIS AND TREATMENT OF APHASIA.

Depends on age of patient and severity of lesion. In the young, recovery may occur, possibly from development of opposite hemisphere. Re-education needs patience. In adults, improvement less common.

II. APRAXIA. AGNOSIA.

A disorder of cerebral functions characterized by inability to perform certain familiar purposive movements, but with absence of motor or sensory paralysis and any general defect of intelligence. The deficiency may be of motor or sensory origin, constituting respectively apraxia and agnosia.

Diseases of the Cerebrum, *continued.*

Apraxia.—Inability to perform a movement corresponding to a correct mental idea, i.e., subject knows what he wants to do, but is unable to do it. Thus motor aphasia is verbal apraxia.

Example.—When given a match, recognizes it as such but unable to strike it. May be bilateral or unilateral (movements correctly performed by one hand). Skilled and complex movements most affected.

PATHOLOGY.—Lesions of the three frontal convolutions; the third (Broca's area) produces motor aphasia, i.e., verbal apraxia, the first and second are probably similar centres for co-ordinating limb movements. Also lesions of anterior portion of corpus callosum (connecting the hemispheres).

Note.—A lesion on the left (in right-handed subjects) may interrupt fibres to the right hemisphere and produce *unilateral left-handed apraxia.*

Agnosia.—Inability to understand meaning of a sensory stimulus. Varieties correspond to various forms of stimuli. Thus sensory aphasia is verbal agnosia; word-deafness is auditory agnosia, latter also including failure to recognize meaning of any sounds; astereognosis is tactile agnosia. Apraxia is necessarily present in agnosia.

Example.—When given a match, does not recognize it as such, may call it a pen.

PATHOLOGY.—Lesions of posterior portion of the external surface of the hemispheres, especially the occipital cortex. Complicated by other deficiencies. Occurs also in diffuse lesions, especially dementia paralytica.

VIII. INTRACRANIAL TUMOURS.

Pathology.—(1) Infective granulomata; (2) Tumours; (3) Cysts.

1. INFECTIVE GRANULOMATA.—

TUBERCLE.—Commonest tumour in childhood, uncommon over 20 years. *Special sites*: Cerebellum, pons. *Size*: Up to golf ball, often multiple; on section cheesy; may be softening. Tuberculosis of bones and glands common; may be terminal tuberculous meningitis.

SYPHILOMA, GUMMA.—Common in adults, rarely in congenital syphilis. *Special sites*: Cortex, pons; is rare in cerebellum. *Origin*: Superficial, from meninges, or arteries. Rarely large, may be multiple; may shrink and become encapsuled. Some gummatous meningitis common at base.

2. TUMOURS.—

GLIOMA.—Commonest tumour in adults; often chronic. *Special sites*: Cerebral cortex, also cerebellum, pons, etc. Consistency varies from firm to soft and vascular, with frequent hæmorrhage. Appearance resembles brain tissue, and tends to infiltrate and not displace substance, hence margin indefinite, and capsule rare. May be a diffuse gliosis in tissues outside the definite tumour.

Histology.—*Origin* usually from neuroglia, occasionally from

ependyma. *Cells*: Vary in different tumours from embryonic cells to neuroglial spider cells and to ganglion cells.

ENDOTHELIOMA, FIBROSARCOMA.—*Special sites*: Cerebellopontine angle, also sagittal sinus. Very chronic; encapsuled; produce pressure effects. Most operable of tumours.

SARCOMA OF BRAIN SUBSTANCE.—Rapid growth. Not common.

CARCINOMA.—Primary rare; secondary not uncommon, especially to breast. Growth rapid. *Special sites*: Cortex, cerebellum, occasionally the choroid plexus.

Other varieties include:—*Fibroma*: from meninges *Osteoma*: from falx cerebri, or growing inwards from cranium. *Psammoma* ('brain-sand'): pineal gland or choroid plexus. *Cholesteatoma*: has glistening appearance, never from brain substance, usually from middle ear in chronic suppuration, and perforates bone. *Lipoma*: from corpus callosum. *Neuroma*: on cranial nerves, especially nervus acusticus. (Von Recklinghausen's neurofibromatosis may also be intracranial.) *Teratoma*: usually pituitary gland. Also *intracranial aneurysms*.

3. CYSTS.—Include: *Porencephaly*: cysts between brain and meninges, from hæmorrhage or maldevelopment dating from birth. *Degenerated tumours*: especially in cerebellum. *Hydatid cysts*. *Cysticercus cellulosæ*, often multiple; produce varied symptoms (*see* CYSTICERCUS).

Etiology.—

SEX.—Males twice as common as females, not accounted for by syphilis.

AGE.—Tubercle, under 20 years. Glioma, 20 to 45 years. Cancer, 40 to 60 years

Symptoms.—Very variable. Two main groups: (A) *General symptoms*: from increased intracranial pressure. (B) *Localizing symptoms*: from irritation and destruction of tissue.

In localization, difficulties may arise from: (1) 'Silent areas'; (2) Increased intracranial pressure affecting nerves distant from tumour; (3) Spreading œdema, meningitis, (4) Distant areas of softening, from tumour compressing vessels.

A. GENERAL SYMPTOMS.—Especially *headache, vomiting, and optic neuritis*.

HEADACHE.—Usually severe and constant, with greater paroxysms. Situation may correspond to tumour, but is not definitely localizing.

VOMITING.—Often early, and persistent; especially in cerebellar and pontine tumours. No nausea, abdominal pain, or relation to food.

OPTIC NEURITIS ('*choked disc*', *papillitis*).—Must be looked for, as vision is often normal until *atrophy* follows later. In tubercle least, in glioma most frequently. *Absence does not negative tumour*: present in about 80 per cent of all tumours. (*See* p. 827.)

VERTIGO.—Common; especially with cerebellar tumours.

MENTAL CHANGES.—Not uncommon in some degree, especially with tumours of frontal lobes: unusual actions, stupor,

Intracranial Tumours—Symptoms, *continued.*

mental dullness; or psychical changes, becoming emotional or hysterical. Mania rare.

CONVULSIONS.—In tumours affecting cortex; rarely elsewhere. May be generalized, Jacksonian, or as in true epilepsy.

SLOW PULSE.

General nutrition only affected late. Polyuria, glycosuria, albuminuria occasionally.

LOCALIZING SYMPTOMS.—(*See also* respective CRANIAL NERVES, when these are referred to.)

PREFRONTAL REGION.—Usually mental dullness and apathy, or emotional changes; may be finally delusions, or dementia. Exophthalmos may develop. Grainger Stewart adds two symptoms: (*a*) Fine tremor of limbs on same side; (*b*) Superficial abdominal reflexes diminished on opposite side. *Extension* occurs into motor or speech area.

MOTOR AREA.—*Ascending frontal convolution.* Irritation at first causes spasm of muscles; destruction of tissue follows, and causes paralysis. (1) *Spasm or convulsions (Jacksonian epilepsy).* Commences in group of muscles of area irritated, and spreads to others. Note: (*a*) 'Signal symptom', i.e., site of commencement, often with tingling; (*b*) March of spasm; (*c*) Subsequent (transient) paresis. (2) *Paralysis.* Commences as monoplegia, e.g., leg, and is permanent and progressive.

Exact localization depends on arrangement of the centres, e.g., face in lower third, upper limb in middle third, lower limb in upper third. On left side also speech centre in Broca's area, 3rd frontal convolution.

Subcortical lesions.—Paralysis occurs first, spasms later as tumour reaches cortex. Sensory symptoms not uncommon, from proximity of tracts.

PARIETAL REGION.—*Ascending parietal convolution.* Impairment of sensation on opposite side of body; especially *light touch*, also of stereognosis.

Extension of tumour may involve: (1) Motor area: Jacksonian epilepsy commencing with local tingling. (2) Supramarginal and angular gyri: word-blindness and mind-blindness (on left). (3) Temporal lobe: word-deafness.

(In the ascending parietal region, sensation is probably represented in local areas opposite to, and connected with, the corresponding motor areas.)

TEMPORAL LOBE.—Mostly consists of 'silent areas' producing no localizing symptoms.

First temporal convolution.—Word-deafness (on left). With destruction, incomplete deafness of *opposite ear*; may be various auditory sensations.

Extension may involve the motor area.

Uncinate gyrus.—Disagreeable subjective sensations of taste and smell. '*Uncinate fits*': (*a*) Attacks of

perversions of taste and smell; (*b*) Jackson's 'dreamy state' of unreality, or of previous identical occurrence of present surroundings.

OCCIPITAL LOBE.—May be latent. *Disturbances of vision* common: (1) Limitation of fields of vision, (i) for colours (usually earliest change), (ii) sight, e.g., homonymous hemianopia: when *cuneus* affected, quadrantic hemianopia occurs (*see also* OPTIC NERVE). Until examination, changes often unsuspected, owing to normal central vision. (2) Visual hallucinations, e.g., coloured scotoma; not common.

Extension may involve: (1) Internal capsule: hemiplegia, hemianæsthesia, hemianopia. (2) Angular gyrus: word-blindness. (3) Cerebellum: ataxia on same side.

INTERNAL CAPSULE.—The closeness of the tracts in the genu and posterior limb results in widespread paralysis. *Order of tracts* (from before backwards): (1) *Genu*: eye, head, tongue, mouth. (2) *Posterior portion, anterior two-thirds*: shoulder, elbow, wrist, fingers, thumb, trunk, hip, ankles, knee, toes. (3) *Posterior portion, posterior third* (retrolenticular): sensory fibres, and finally *optic radiations*. The motor tracts are most commonly affected, and of these the *face least*. The *localizing symptoms* are thus: *hemiplegia, hemianæsthesia, hemianopia, without convulsions*. *Aphasia* only occurs with bilateral affections ('pseudobulbar paralysis').

BASAL GANGLIA.—Small tumours may cause no localizing symptoms. By extension, involve the internal capsule at different sites, causing paralyses.

OPTIC THALAMUS.—*Thalamic syndrome** (Roussy): (1) Persistent *hemianæsthesia*, especially to deep sensibility, but also to touch, pain, and temperature. (2) Slight transient hemiplegia without contractures. (3) Hemiataxia (slight) and astereognosis. (4) *Severe, persistent, and paroxysmal pains* on affected side. (5) *Tremor*, or *choreic* or *athetotic movements*, on affected side.

CORPORA QUADRIGEMINA.—(1) *Disturbance of equilibrium*, causing reeling gait. (2) *Ocular symptoms*: nystagmus, loss of pupil reflexes. *Crus* usually involved, whence: (3) *3rd nerve paralysis*, especially *ptosis*. (4) Crossed hemiplegia. *Anterior body* is connected with visual tracts, and *posterior body* with auditory tracts; if latter affected, hearing liminished, especially on opposite side.

CRUS.—'*Crossed paralysis*': (1) 3rd nerve on same side. (2) Hemiplegia on opposite side. May also be hemianæsthesia, if fillet affected; lesions of 4th and 6th nerves.

[*Tegmentum*.—If the nuclei ruber, and the connecting superior cerebellar peduncle (the cerebellorubral

* Investigated by Head and Gordon Holmes. *Croonian Lectures*, 1911.

Intracranial Tumours—Symptoms, *continued.*

system), be affected, a syndrome occurs: (1) Coarse tremor; (2) Loss of emotional movements of face; (3) Ataxia. (Gordon Holmes.)]

PONS.—Tubercle and glioma are not uncommon; glioma may become large without causing localizing symptoms, by surrounding, without destroying, nerve fibres. Optic neuritis also unusual or late. Symptoms very variable.

'Crossed paralysis' usual, many variations: (1) *6th and 7th nerves and pyramidal tract*; whence (a) facial paralysis on side of tumour, (b) conjugate deviation of eyes to opposite side (*see* SIXTH CRANIAL NERVE), (c) hemiplegia of limbs on opposite side. (2) *5th nerve and pyramidal tract*; whence (a) anæsthesia of face on same side, (b) complete hemiplegia on opposite side. (3) In addition to last, the fillet may be affected whence anæsthesia of limbs on opposite side to face, i.e., *crossed sensory paralysis* (the motor tracts may escape).

Note.—Tumours of pons may also involve 6th nerve below its nucleus, affecting the rectus externus only, not causing conjugate deviation.

Extension of tumour may cause: (1) Bilateral symptoms, common; combinations of above paralyses. Also anarthria. (2) Involvement of cranial nerves, e.g., 8th (deafness). (3) Involvement of medulla (dysphagia). (4) Involvement of middle cerebellar peduncles (ataxia). (4) Distention of ventricles.

MEDULLA.—Primary tumour very rare. Affects: (1) Cranial nerves, 9th, 10th, 11th, and rarely 12th. Difficulty in articulation and swallowing; irregular heart and respiration. (2) Motor tracts: hemiplegia. By *extension*, usually also cerebellum and pons.

TUMOURS OF CEREBELLO-PONTINE ANGLE TUMOURS OF NERVUS ACUSTICUS. (Also known as 'extracerebellar' tumours. *See* Grainger Stewart and Gordon Holmes, *Brain*, 1914, xxvii, 522.)—Not uncommon. Circumscribed and encapsuled. Localization often definite. Operative removal frequently possible. *Origins* (1) From cranial nerves, especially *sheath of auditory nerve*; pathology doubtful (? fibromyxoma). (2) Surface of cerebellum, less commonly; usually glioma.

General symptoms absent or late.

Localizing symptoms from compression of (1) cranial nerves, (2) cerebellum, (3) pons.

1. Cranial nerves: *Earliest symptoms* on side of tumour. Order of affection: (i) 8th nerve: nerve deafness, becoming complete, also tinnitus. (ii) 7th nerve: *slight* facial paralysis. (iii) 6th nerve: external rectus only. (iv) 5th nerve: tingling in area.
2. Cerebellum: Homolateral ataxia, paresis, and atonia. Vertigo. Nystagmus.

3. Pons: *Hemiplegia on opposite side*, usually slight (contralateral spastic paralysis).

Diagnosis from intracerebellar tumours. (1) *Nerve deafness* on side of tumour: tinnitus. Also 5th nerve. (2) Paresis of limbs on opposite side. (3) Plantar reflex often extensor. (4) Vertigo: sensation of *rotating towards* side of lesion (opposite in cerebellar tumours).

In pontine tumours note: crossed paralysis, conjugate deviation, sensory changes, indefinite vertigo.

In tumours from base of skull, posterior fossa, note: deafness less complete, pain in 5th nerve more marked, cerebellar symptoms later. Often very difficult.

In labyrinthine disease. Bárány's tests, e.g., symptoms increased by injection of hot water into external auditory meatus; also other tests.

TUMOURS ARISING FROM BASE OF SKULL.—Usually sarcoma. May perforate into nasopharyngeal cavity or orbit. *Symptoms* mainly by compression of cranial nerves; general symptoms often late. *From anterior fossa*: nerves of eye affected, blindness and ocular paralysis, may be protrusion of eyeball (orbit invaded); anosmia, mental changes (frontal lobes). *Middle fossa*: especially 5th nerve (pain, impaired sensation, inflammation of eye, wasting of masseter); by extension of tumour, 7th, 8th and ocular nerves, uncinate gyrus. *Posterior fossa*: 5th, 6th, 7th, and 8th nerves, later pons and cerebellum.

PITUITARY GLAND.—*See* p. 753.

CEREBELLUM.—*See* p 875.

Diagnosis.—*Questions arising* are: (1) Is a tumour present? (2) Where is it situated? (3) What is its character?

1. PRESENCE OF TUMOUR.—Mainly decided by general symptoms, headache, vomiting, and optic neuritis. Difficulties from:—

   i. NEPHRITIS, URÆMIA, AND SPREADING ŒDEMA OF BRAIN.—Similar syndrome occurs and retinitis may be absent. Albuminuria and casts present. Albuminuria may be scanty (chronic interstitial nephritis), but if so and above symptoms present, arteriosclerosis is always advanced.

   ii. INTRACRANIAL ABSCESS.—Note: primary focus, pyrexia, signs of sepsis, leucocytosis. Choked disc very rare.

   iii. HYSTERIA.—Early tumours may simulate hysteria.

   iv. DEMENTIA PARALYTICA.—Occasional confusion with tumours of frontal lobe.

   Also occasionally: lead encephalopatny, cerebral vascular lesions, local meningitis, hydrocephalus. In hypermetropia, headache, vomiting, and congestion of discs may occur.

2. SITUATION OF TUMOUR.—*See* LOCALIZING SYMPTOMS.

3. CHARACTER OF TUMOUR.—(Wassermann reaction at earliest moment.) Tubercle: especially in children and in cerebellum. Syphiloma: usually on cortex, hence convulsions. Decision usually impossible.

Intracranial Tumours, *continued.*

Course and Prognosis.—Symptoms slowly progress. Paralyses occurring are permanent. *Duration of life* from onset of symptoms rarely exceeds two years.

SYPHILOMA.—Only curable tumour.

GLIOMA.—Rarely, duration ten or more years.

TUBERCLE.—Rarely, may become quiescent.

PROGNOSIS BAD.—With rapid optic neuritis, persistent vomiting or convulsions, definite mental symptoms.

DEATH.—Occurs: (1) From coma; (2) From exhaustion, headache, and vomiting; (3) Suddenly, tumours usually affecting medulla. Occasionally from meningitis, generalized tuberculosis, secondary growths, hæmorrhage, etc.

Treatment.—

A. MEDICAL.—In *syphiloma*, antisyphilitic treatment (*trephining previous* to treatment must be considered). General medical treatment palliative in other forms; iodides may give transient relief. *Headache*: ice-bags, phenacetin; but if severe needs morphia. *Convulsions*: bromides of little use.

B. SURGICAL.—To be considered immediately on diagnosis.

1. REMOVAL.—Decision rests on localization in accessible site, and condition of patient. Complete recovery in very small percentage. Mortality high from shock, meningitis, etc. Cerebral cortex and cerebellum most accessible. Tumours of cerebellopontine angle most easily removable, but shock marked and mortality high.
2. TREPHINING: DECOMPRESSION.—Palliative treatment. (1) Relieves headache and persistent vomiting, and often convulsions; (2) Relieves optic neuritis (often subsides rapidly), and prevents subsequent atrophy and blindness.

IV. ABSCESS OF THE BRAIN.

Etiology.—Probably always secondary.

TRAUMA.—Not uncommon. Abscess usually at site of injury.

EXTENSION OF LOCAL INFLAMMATION.—Commonest cause. Foci: (1) *Middle-ear disease*, usually chronic; spreads by caries through roof of the tympanum or by vessels. *Site of abscess*: commonly temporal lobe. (2) *Mastoid-cell suppuration*. *Site*: commonly cerebellum. Sinus thrombosis frequent. Less commonly: (3) *Frontal and accessory nasal sinuses*. *Site*: frontal lobe. (4) Syphilitic and tuberculous caries of bone. (5) Facial erysipelas, carbuncle, etc. (rare).

DISTANT SEPSIS.—Unusual cause. (1) Pulmonary sepsis, e.g., bronchiectasis, pulmonary abscess. (2) Pyæmia and infective endocarditis. Small multiple abscesses may occur. (3) Influenza, enteric fever: rarely. Rarely in: empyema, sepsis of liver or bones. *Site of abscess* depends on origin: (1) *Temporal lobe* most common, especially 3rd convolution; (2) Cerebellum.

Morbid Anatomy.—Usually single, except in general pyæmia. *Size*: often about that of walnut.

ACUTE ABSCESS.—Not definitely limited; surrounding œdema.
CHRONIC ABSCESS.—Often definite capsule. Contains green pus with offensive odour (probably anaerobic organisms).
BACTERIOLOGY.—Various micrococci and bacilli. May be sterile. Path of infection not always recognizable

Symptoms.—Vary greatly according to site of abscess, symptoms of primary disease, and of ancillary disease, e.g., sinus thrombosis, meningitis.

COURSE may be: (1) *Acute*, especially after injury; duration two to three weeks. (2) *Latent*: may be several months (also occurs after injury). (3) *Chronic*, especially in ear group.

STAGES may be recognizable: (1) *Invasion*, headache and malaise; (2) *Latent*; (3) *Terminal*, due to (*a*) spreading inflammatory œdema, or (*b*) rupture of abscess causing meningitis, or into ventricles.

Symptoms resemble rapidly-growing tumour: (*A*) General; (*B*) Localizing.

A. GENERAL SYMPTOMS.—Less marked than in tumours.

HEADACHE.—Rarely absent.
OPTIC NEURITIS.—Less frequent and marked than in tumour.
VOMITING AND VERTIGO.—Mainly in cerebellar abscess.
MENTAL CHANGE usual. Drowsiness and apathy. Later, stupor and coma.
PULSE.—Often slow. In terminal stage, rapid or irregular.
TEMPERATURE.—In latent and uncomplicated forms, is usually normal or subnormal. Rises in terminal stage; high with sinus thrombosis or rupture into ventricles.
RESPIRATION often slow, especially in cerebellar abscess.
ANOREXIA, furred tongue, and some septic absorption not uncommon.
LEUCOCYTOSIS.—Often marked, but not invariable.
In 'acute' forms, signs of sepsis and meningeal irritation more definite: pyrexia, irritability or delirium, rigors.

B LOCALIZING SYMPTOMS (*see* INTRACRANIAL TUMOURS for details).—Accurate localization usually not possible. Of special sites:—

TEMPOROSPHENOIDAL ABSCESS.—May be: (1) *Deafness* on opposite side. (2) Taste and smell affected, rarely. (3) Incomplete *word-deafness* (if on left). (4) Superficial abdominal reflexes lost on opposite side. *Pressure effects* when growing: (*a*) downwards, 3rd and 6th nerve; (*b*) inwards, internal capsule; (*c*) angular gyrus, word-blindness, hemianopia; (*d*) sensory and motor cortex.
CEREBELLAR ABSCESS.—General symptoms marked; nystagmus to side of lesion. *Lies on opposite side*, with eyes also away from lesion. Ataxia and paresis on side of lesion. Reeling gait.

Diagnosis.—General considerations suggesting abscess: (1) *Symptoms of tumour with pyrexia*; (2) Cessation of discharge in chronic otitis media; (3) Suggestive symptoms following injury, or in

Abscess of the Brain—Diagnosis, *continued.*

presence of other etiological factors. In doubtful cases these etiological factors must be sought for to ascertain presence at the time or their history in the past.

SPECIAL DIAGNOSIS from: (1) *Mastoiditis.* Drowsiness and optic neuritis suggest abscess. (2) *Meningitis.* Usually definite pyrexia, irritability, photophobia, and, may be, convulsions. Cerebrospinal fluid under pressure; may contain pus-cells and micro-organisms. (Septic meningitis may follow otitis media and co-exist with abscess.) (3) *Sinus thrombosis.* Usually abrupt onset, high temperature, rapid pulse, rigors, with swelling and tenderness at exit of internal jugular vein. May co-exist with abscess. (4) *Intracranial tumour.* Progress more gradual; optic neuritis usually early and marked; no leucocytosis; no etiological factors of abscess.

Treatment.—*Operation and drainage* at earliest moment. Subsequent mortality occurs from: (1) Meningitis and encephalitis. (2) Exhaustion, especially in late operations. (3) Sinus thrombosis and general septicæmia. (4) Multiple abscesses. (5) Abscess insufficiently drained. (6) Pulmonary disease and other causal conditions. In absence of operation, invariably fatal.

V. CEREBRAL PALSIES OF CHILDREN.

Characterized by paralyses of upper motor neuron type with certain accessory symptoms. Cause may arise at, after, or (probably) before birth. Main types are hemiplegia and diplegia.

1. INFANTILE HEMIPLEGIA.

Etiology.—Onset from birth or under 2 years. Rare over 5 years.

Causes.—(1) *Injury at birth,* especially from forceps: meningeal hæmorrhage, or other injury to cortex. (2) *Acute polio-encephalitis*: cerebral equivalent of acute poliomyelitis. (3) Hæmorrhage, thrombosis, and embolism as in adults. Rare, and usually in the older children. Bleeding commonly from veins, e.g., pertussis, severe convulsions.

Morbid Anatomy.—Gross changes, examined long after onset, are: (1) *Atrophy and sclerosis of brain.* Usual form, especially from acute encephalitis. Area, from small portion to entire hemisphere. Meninges adherent, brain substance hard. (2) *Porencephaly,* viz., cysts on surface with deficiency of brain tissue, may communicate with ventricles. Origin may be (*a*) hæmorrhage at birth, (*b*) possibly defective development.

Symptoms.—

ONSET IN ACUTE ENCEPHALITIS.—Sudden. Age usually 2 to 5 years. Symptoms: (1) *Pyrexia*; (2) *Loss of consciousness*—few hours to days; (3) *Convulsions,* local or general—occasionally absent.

HEMIPLEGIA.—Noticed on return of consciousness; may be partial at first and extend with recurring convulsions; commoner on right side.

RESIDUAL SYMPTOMS (for all forms).—

1. PARALYSIS.—May recover almost completely, especially face and leg more rapidly than arm. '*Residual paralysis*' common: (i) Hemiplegic gait; (ii) Upper limb flexed at elbow and wrist, fingers flexed at metacarpo-phalangeal joint, and extended at others. *Reflexes* increased. *Sensation* unchanged. *Arrest of development*: affected limbs smaller, face may be asymmetrical.
2. MENTAL DEFECTS.—Common. All grades from 'backwardness' to idiocy. *Aphasia*: not uncommon if onset when child can speak; if earlier, may learn to use opposite hemisphere.
3. INVOLUNTARY MOVEMENTS.—Common. (i) *Post-hemiplegic chorea*: vary from tremors to severe choreiform movements. (ii) *Athetosis*: slow, involuntary, more or less rhythmical movements of extremities, usually of fingers, from position of supination, extension, and abduction, to pronation, flexion, and adduction.
4. EPILEPSY.—Common: *petit mal*, Jacksonian, or general convulsions. Distribution extends with repetition.

Prognosis.—*Paralysis* often improves to an unexpected degree. Bad features are: (1) Mental defects (influencing treatment). (2) Athetosis. (3) Epilepsy: tends to produce, or increase, mental defect.

Treatment.—

FEBRILE STAGE.—Bed Purge. Ice to head. Chloroform if recurrent convulsions.

RESIDUAL PARALYSIS.—Indications: to maintain nutrition of muscles and prevent contractures. *Massage* over long periods *Exercises*, active and passive.

CONTRACTURES.—Treatment by operation (tenotomies, etc.) and apparatus.

EPILEPSY.—Bromides usually fail; borax recommended.

MENTAL DEFECTS.—Long and careful education necessary.

2. CEREBRAL DIPLEGIA.

(*Little's Disease. Spastic Paralysis of Infants.*)

Etiology.—Practically always present from birth. Often first or difficult labours.

Causes.—(1) *Injury at birth.* Hæmorrhage possibly from longitudinal sinus or veins. (2) *Defective development* of motor cortex and tracts.

Morbid Anatomy.—Changes may be: (1) Atrophy and sclerosis; (2) Commonly *porencephaly*.

Symptoms.—Often first noticed from delay in walking or sitting up. *Convulsions* may occur in infancy.

1. PARALYSIS AND RIGIDITY.—*Legs* more affected than arms; *spasm of adductor muscles of thighs*, knees flexed, heels drawn

Cerebral Diplegia—Symptoms, *continued.*

up by calf muscles; hence, when held up stands on toes and inner side of feet, or legs crossed; may be 'scissors gait'. *Arms*: deficiency often slight; may be flexed joints. *Face* often escapes, or involuntary grimaces occur. *Reflexes* increased; plantar reflex extensor (*N.B.*—Normally so in infants). *Sensation* normal No wasting.

2. MENTAL DEFECTS.—All grades. (*See* INFANTILE HEMIPLEGIA.)
3. INVOLUNTARY MOVEMENTS.—Various. Spastic movements when attempting to seize objects. Also 'post-hemiplegic chorea' and 'athetosis' (*see* INFANTILE HEMIPLEGIA), may be bilateral and very extensive.
4. EPILEPSY AND CONVULSIONS.

Diagnosis.—Simple. May resemble syphilitic meningo encephalitis.

'CONGENITAL SPASTIC IDIOCY'.—Applied to type with dementia and slight rigidity.

'LITTLE'S DISEASE'.—Especially applied to paraplegia.

Prognosis and Treatment.—*See* INFANTILE HEMIPLEGIA.

CEREBELLAR PALSIES.

Etiology.—Resembles cerebral palsies.

Groups (Batten).—(1) *Congenital cerebellar ataxia.* From birth (cf. CEREBRAL DIPLEGIA). (2) *Acute cerebellar ataxia.* Onset from birth, or following acute fevers or encephalitis. These forms tend to improve. (3) Progressive cerebellar ataxia.

VI. HYDROCEPHALUS.

Properly, any accumulation of serous fluid within the cranium. (*a*) *Hydrocephalus externus,* fluid between cortex and skull. Occurs in atrophy of brain, old age, general wasting, haemorrhage, etc. Not further referred to here. (*b*) *Hydrocephalus internus,* increase of fluid in the ventricles.

Congenital Hydrocephalus.—May be present at birth and obstruct labour. Most commonly cranial enlargement commences subsequently. Pathology unknown: ventricular foramina not always obstructed; sometimes congenital syphilis. Spina bifida may co-exist.

GENERAL DESCRIPTION.—*Skull* enormously enlarged, with face of normal size. Bones thin, sutures wide, Wormian bones numerous. Ventricles greatly distended, and brain substance very thin. Fluid clear. Ependyma may be granular, but signs of inflammation rare; also in choroid plexus.

SYMPTOMS.—Convulsions, general spasticity, and increased reflexes common. Mental condition: various degrees of deficiency, rarely normal.

Death usually within three or four years.

Acquired Chronic Hydrocephalus.—From interference with cerebrospinal circulation, especially: (1) *Tumour* obstructing veins of Galen, e.g., cerebellopontine or cerebellar. (2) *Sequel*

of meningitis (especially cerebrospinal) blocking foramen of Magendie.

SYMPTOMS variable. Headache, slow pulse, gradual blindness, mental changes; may be attacks of coma of long duration. May resemble tumour.

Idiopathic Internal Hydrocephalus (*Quincke's serous meningitis*).—Rare condition, occurs in children or adults. Ascribed to ependymitis producing serous effusion into ventricles; symptoms, resulting from distention, resemble meningitis or tumour. Cause unknown.

ACUTE.—Resembles meningitis: headache, head retraction, slow pulse, optic neuritis. No fever.

CHRONIC.—Resembles tumour. Headache, optic neuritis, etc.; may be drowsiness or coma of several weeks' duration. Convulsions and cranial nerve paralysis.

PROGNOSIS.—Recovery may occur. Accounts for some apparent recoveries from intracranial tumour and meningitis.

Treatment of Hydrocephalus.—Numerous operations for draining the ventricles have been tried, but none is satisfactory.

VII. AMAUROTIC FAMILY IDIOCY.

(*Tay-Sachs' Disease.*)

A fatal disease of infants characterized clinically by progressive mental, motor, and visual failure, and by pathognomonic changes in the retina; and pathologically by swelling of the cytoplasm of the cells of the central nervous system.

Etiology.—Symptoms appear between 3rd and 6th month. *Familial factor* marked; almost, if not entirely, confined to Hebrews. No relation to syphilis, or trauma at birth: consanguinity may be present.

Morbid Anatomy.—Characteristic *change in cells of nervous system*: protoplasm greatly swollen and cellular network absent, *cells presenting 'ballooned' appearance*, with nucleus pushed aside and often destroyed; dendrites swollen, but axon little affected. Widespread, and few cells escape in cortex, ganglia, or cord. No signs of inflammation; neuroglial proliferation slight and secondary. May be some atrophy and sclerosis of convolutions.

Ganglion cells in retina similarly affected and cause of specific appearance at macula.

Pathogenesis.—A congenital defect and degeneration of the cell protoplasm.

Symptoms.—Infant appears normal at birth and until three to six months.

INITIAL SYMPTOMS.—Weakness of neck and back muscles, unable to sit up, head falls forward. Vision defective.

PROGRESS AND CHARACTERISTIC SYMPTOMS.—

1. IDIOCY develops.
2. PARALYSIS becomes complete; unable to move; wasting extreme.

Amaurotic Family Idiocy—Symptoms, *continued.*

3. BLINDNESS becomes total.
4. PATHOGNOMONIC BILATERAL RETINAL CHANGE.—At macula an oval white area, larger than the optic disc, with a central 'cherry red spot' (fovea). Also optic atrophy

Spasticity may, or may not, develop, with increased reflexes, spasms and contractures, but never marked. No convulsions. No sensory changes.

Occasionally: Slow lateral nystagmoid movements. Hyperacusis.

Course.—*Fatal* at 1½ to 2½ years.

Diagnosis.—By family history, progressive mental, motor, and visual changes, absence of convulsions, and by pathognomonic appearance of retina.

Treatment.—Palliative. Weaning useless (Sachs).

[A juvenile type, onset between 6 to 14 years, is at present unconfirmed.]

VIII. PROGRESSIVE LENTICULAR DEGENERATION.

Recognized and described by Kinnier Wilson (*Brain*, 1904 and 1912).

Morbid Anatomy.—Two main lesions: (1) Bilateral softening of the lenticular nucleus, especially the putamen. (2) Cirrhosis of the liver: not identical with ordinary types.

Etiology.—*Onset in youth. Familial,* but not hereditary or congenital.

Symptoms.—Characteristics: (1) Tremors; (2) Muscular weakness, spasmodic contractions, spasticity, and contractures; (3) Difficulty in articulation and swallowing; (4) Emotional and often mental changes.

Course.—Progressive, with emaciation. Fatal. *Duration*: a few years. No obvious symptoms from the cirrhosis.

CHAPTER CXXXI.

VASCULAR LESIONS OF THE BRAIN.

I. CEREBRAL HÆMORRHAGE.

(*Apoplexy.*)

Etiology (factors associated with degeneration of vessels).—

AGE.—Especially 40 to 60 years; rarely under 40.

SEX.—*Males* common (from predisposing factors).

HEREDITY.—Familial tendency to vascular degeneration occurs Also plethoric build.

PREDISPOSING FACTORS.—(1) *Chronic interstitial nephritis and causes of arterial degeneration and high blood-pressure*, viz., alcohol, over-eating, syphilis, chronic muscular strain, gout, and lead. Cardiac hypertrophy common. Other occasional causes: (2) Infective endocarditis, with embolism and aneurysm. Rarely: (3) Acute specific fevers. (4) Temporary high blood-pressure, e.g., whooping-cough paroxysms, parturition. (5) Anæmia. Also: (6) Birth injuries.

EXCITING CAUSES.—May be none obvious, occurrence not uncommon during sleep. Events affecting circulation. e.g., emotion, muscular strain (e.g., in constipation).

Pathology.—

VESSELS OF ORIGIN.—Commonest are branches of middle cerebral artery through anterior perforated space, (specially: (1) *Lenticulo-striate artery* of Duret (the artery of hæmorrhage): pierces base of brain, enters external capsule, ascends between this and lenticular nucleus, then through the latter and the anterior portion of the internal capsule, finally ending in the caudate nucleus. (2) Lenticulo-optic branches: supply posterior (retro-lenticular) portion of internal capsule. Frequency of rupture ascribed to: (a) Origin at right angles to middle cerebral; (b) Absence of anastomosis ('end arteries').

MORBID HISTOLOGY.—Hæmorrhage arises from:—

1. *Miliary aneurysms.* Commonest lesion. Often numerous, size of pin's head. Especially on branches through anterior perforated space. Origin doubtful, may occur without degeneration in larger arteries.
2. Aneurysms of circle of Willis.
3. Degeneration of cerebral vessels without aneurysms.
4. Hæmorrhage into soft tumours.
5. Diapedesis: possibly cause in acute infectious diseases and anæmia.

SITE OF HÆMORRHAGE.—

INTRACEREBRAL HÆMORRHAGE.—(1) *Internal capsule*: commonest site. (Often commences external to capsule.) Lenticular nucleus and optic thalamus may be involved. (2) *Pons.* (3) Less commonly: cortex, centrum semiovale, cerebellum, crus, temporo-sphenoidal lobe.

MENINGEAL HÆMORRHAGE.—May be extra- or intradural. Occurs in: (1) Fractures and head injuries. (2) Aneurysms—usually middle meningeal artery. (3) Birth injuries. (4) Occasionally: acute infectious fevers, anæmia, extension of intracerebral hæmorrhage. Effusion may be large and flow to base and spinal cord.

INTRAVENTRICULAR HÆMORRHAGE.—Rarely primary. Usually extension from intracerebral hæmorrhage. Tends to flow into opposite ventricle, and also into 3rd or even 4th ventricle.

SUBSEQUENT CHANGES IN THE HÆMORRHAGE.—The effused blood darkens in colour. Subsequently, either: (1) Formation of a wall enclosing a fluid cyst; or (2) Absorption of

Cerebral Hæmorrhage—Pathology, *continued.*

the blood, proliferation and organization of connective tissue, leaving a pigmented scar. Brain tissue around shows staining.

In meningeal hæmorrhage, blood may be absorbed. With birth hæmorrhages, when profuse, cortex may waste and cysts form (porencephaly).

SECONDARY DEGENERATION OF NERVE FIBRES affected occurs, and can be traced in the tracts involved.

Symptoms.—Characteristics are: (a) Initial phenomena: unconsciousness or coma, 'apoplectic fit'. (b) Paralysis.

PREMONITORY SYMPTOMS OR 'WARNINGS'.—Probably from small hæmorrhages. Frequently absent. Numbness or tingling in limbs; attacks of headache, giddiness, or vomiting; epistaxis; disturbances of vision, retinal hæmorrhages; slight difficulty in speaking or mental disturbance; slight convulsions and choreiform movements.

TYPES OF ONSET.—Vary with the extent and position of the hæmorrhage.

SUDDEN LOSS OF CONSCIOUSNESS.—Not common.

GRADUAL DEVELOPMENT OF PARALYSIS AND COMA.—From few minutes upwards. Common form. The longer onsets termed 'ingravescent apoplexy'.

PARALYSIS WITHOUT COMA.—From small central hæmorrhages.

COMA.—Occurs of any depth. May be able to put out tongue, or attempt to speak. Rapid and deep in small hæmorrhages in pons, or large effusions anywhere; marked when intraventricular.

CONVULSIONS.—Not common. Most in pontine and cortical hæmorrhages.

APOPLECTIC ATTACK.—*General description* (especially refers to capsular hæmorrhage):—

DEEP COMA.—Rotation of head and eyes common, towards lesion. Face cyanotic, or congested. Respirations slow and noisy, often irregular; lips splutter; cheeks blown out. Pupils inactive, usually dilated; may be unequal; contracted if pontine. Pulse slow, full, incompressible. Temperature normal or subnormal; high if pontine. Reflexes absent. Incontinence of urine and fæces. If hemiplegia: (1) Loss of tone in affected muscles, limbs drop dead; (2) Paralyzed cheek blown out on expiration; (3) Chest movement diminished.

Note.—All muscles may be flaccid during coma.

WHEN ONSET LESS ABRUPT.—Gradual loss of power and unconsciousness passing into deep coma. Premonitory symptoms may precede onset. May occur in sleep, and patient may awake paralyzed, or be found unconscious. *Paralysis without unconsciousness* may occur with small hæmorrhages in internal capsule.

SUBSEQUENT COURSE WHEN COMA DEEP.—May be: (a) *Death* in few hours, very rare under an hour. (b) *Consciousness recovered*, then relapse into fatal coma; more frequent, especially when hæmorrhage bursts into ventricles. (c) *Con-*

sciousness recovered, usually in about twenty-four hours; passes through *stage of febrile reaction* and early rigidity; subsequently symptoms due to hemiplegia.

CONJUGATE DEVIATION (for mechanism, *see* SIXTH NERVE).—Both eyes and often head rotated to one side: (1) Towards lesion, if this is between cortex and crus; (2) Away from lesion, if in pons. In 'early rigidity' the directions are reversed owing to spasm of muscles.

REFLEXES.—During coma, all reflexes absent; with consciousness they return on unaffected side. On paralyzed side the reflexes gradually return, then become *increased*; *plantar reflex extensor* ('positive Babinski's sign'); *ankle-clonus present*. Superficial reflexes diminished.

STAGE OF FEBRILE REACTION.—'*Early rigidity*'. Due to inflammation around hæmorrhage and absorption of blood. Onset 12 to 48 hours after attack.

Temperature rises, headache and malaise. Duration, one to several weeks. 'Early rigidity', stiffness of paralyzed limbs. Trophic changes, e.g., sloughing at lower part of back, in serious cases. Congestion of base of lungs frequent. Difficulty in speech and mental disturbances common for some days. Sphincters unaffected.

HEMIPLEGIA.—From destruction of motor cortex or pyramidal tracts.

GENERAL CHARACTERISTICS OF PARALYSIS.—Hemiplegia partial or complete, i.e., face, arm, and leg. Initial distribution wider than later, from œdema around lesion and irritation of tracts not destroyed. Extent lessens as site of lesion approaches cortex (rapidly above capsule). *Muscles used symmetrically escape*, especially thorax and abdomen (stimulated from either hemisphere). Muscles used in specialized movements suffer most, e.g., arm more than leg, hand more than shoulder. *Face*: paralysis is partial, frontalis and orbicularis palpebrarum escaping (*see* SEVENTH NERVE); tongue and palate affected; emotional movements suffer less than voluntary. Paralysis of upper motor neuron type (except in crossed paralysis). Psychic disturbance common. Sphincters unaffected. Difficulty in speech or, with right hemiplegia, aphasia may occur. Occasional symptom: *pain in limbs*: infrequent: rarely severe (? optic thalamus lesion).

ORDER OF RECOVERY.—Inverse to frequency and severity of affection, viz., leg before arm, shoulder before hand, thumb last. Face may recover rapidly.

RESIDUAL PARALYSIS.—Tends to involve: (a) *Leg*: flexors of legs and dorsiflexors of foot, i.e., shorteners of leg used in second stage of walking. (b) *Arm*: muscles opening the hand and rotating the arm outwards.

LOCALIZING SYMPTOMS (*see also* CEREBRAL TUMOURS, p. 854).—(Commonest site internal capsule, then pons.)

CORTEX.—Paralysis usually limited, but permanent. May be convulsions at onset, aphasia.

CORONA RADIATA.—Paralysis usually limited.

Cerebral Hæmorrhage—Symptoms, *continued.*

INTERNAL CAPSULE.—Paralysis is often widespread and permanent. Site of lesion is revealed by extent and distribution of paralysis Usually in anterior two-thirds of posterior limb. If in posterior third, hemi-anæsthesia and homonymous hemianopia occur.

CRUS.—*Crossed paralysis:* (1) Third nerve on same side; (2) Face and limbs on opposite side. Hæmorrhage often an extension from internal capsule and then involves fillet, whence anæsthesia on paralyzed side Special senses or optic tract may be affected (hemianopia).

'*Crossed Paralysis or Hemiplexia*'.—A cranial nerve affected on one side (lower motor neuron), and a hemiplegia on the opposite side. Occurs in lesion of crus, pons, and medulla, due to levels of decussation.

PONS.—General symptoms at onset: (a) *Pyrexia*, often 105°; (b) *Pupils contracted*; (c) Convulsions not uncommon; (d) *Coma* sudden, deep, and may be fatal. Hæmorrhage often affects both sides.

Lower Portion of Pons.—Crossed paralysis: (1) Sixth and seventh nerves, whence (*a*) facial paralysis on side of lesion, (*b*) conjugate deviation away from lesion; (2) Pyramidal tract, whence limbs paralyzed on opposite side to lesion.

Middle of Pons (rare).—Crossed paralysis: (1) Fifth nerve on side of lesion, i.e., loss of sensation; (2) Hemiplegia on opposite side. Fillet may also be involved, i.e., anæsthesia on opposite side (crossed sensory paralysis).

MEDULLA.—Rare: death usually rapid. Crossed paralysis: (1) Twelfth nerve, whence tongue protrudes towards lesion (usually); (2) Pyramidal tract, hemiplegia on opposite side.

VENTRICLES.—Coma marked. Usually return of coma after an initial recovery of consciousness, from secondary rupture of hæmorrhage into ventricles. Always fatal.

CEREBELLUM.—Rare; diagnosis difficult. Usually superior cerebellar artery, branch to dentate nucleus. Onset with *vomiting, pain* in neck or back of head. Usually fatal from rupture into 4th ventricle. If recovery, localizing cerebellar symptoms (*see* CEREBELLUM, pp 874, 876).

SENSORY CHANGES.—Variable. With hemiplegia, usually slight numbing, hemi-anæsthesia rare. Lesion in posterior third of internal capsule (retrolenticular portion) may give: (1) *Hemianæsthesia.* (2) *Hemianopia,* homonymous to opposite side. Rarely, other special senses. (3) *Leg* more affected than arm. Anæsthesia, without special senses affected, when fillet involved (*see* CRUS and PONS).

Note.—*Protrusion of tongue*: usually deviates to paralyzed side (sound geniohyoglossus), but sometimes the reverse; mechanism unknown.

MENINGEAL HÆMORRHAGE.—

TRAUMA.—At onset may be: *convulsions, unequal pupils* (large on side of lesion). Three stages: (1) Unconsciousness;

following injury. (ii) 'Lucid interval': duration few hours to two or rarely four days. May feel well. (iii) Coma develops, spasms, paralysis, and death. Localized by site of spasms. Stages commoner in extradural than subdural forms; in latter coma rapid, and lucid interval rare.

ANEURYSM.—Source, usually middle meningeal artery. At onset: headache, vomiting, giddiness, and convulsions. Rapid coma.

Sequelæ in Hemiplegia.—

1. SECONDARY CONTRACTURES.—'Late rigidity'. Results permanent.
   - ARM.—Flexion at elbow, wrist, and finger.
   - LEG.—Contractures less marked.
   - *Gait.*—Leg, when walking, is swung in semicircle to prevent toes dragging.
2. DEEP REFLEXES increased.
3. ATROPHY OF MUSCLES unusual. May occur as result of secondary changes in ventral horns.
4. TROPHIC CHANGES.—Skin thin and glossy.

OCCASIONAL PHENOMENA:—

- ASSOCIATED MOVEMENTS.—With strong action of sound limb, movement of paralyzed limb may occur. Possibly impulse spreads to opposite side in lower centres.
- ATHEROSIS, POST-HEMIPLEGIC CHOREA, ARTHROPATHIES.—Mainly in children (*see* p. 860).
- *Note.*—Mental powers and concentration usually impaired after apoplexy.

Diagnosis during Coma (*see also* COMA, p. 294).—Obtain if possible: (1) Previous history: prodromata, fits, alcoholism, previous attacks, etc. (2) Mode of onset: injury, drinking, convulsions, rapidity of onset. Examine: (1) Head for injury. (2) Paralysis: cheeks puffed out, limbs flaccid, reflexes, conjugate deviation. (3) Pupils. (4) Heart and pulse. (5) Temperature. (6) Urine.

If paralysis is present, cause is hæmorrhage, embolism, or thrombosis. (For special symptoms, *see* EMBOLISM.) Diagnosis of thrombosis from hæmorrhage often difficult; former specially suggested by extending paralysis with slight or no loss of consciousness.

Other causes of coma include: alcohol, opium or other narcotic drugs, epilepsy, uræmia, diabetes, various convulsions of nervous system, severe hæmorrhage (*see* COMA).

Alcohol, injury, and hæmorrhage often co-exist; diagnosis uncertain and catastrophes common; treat all doubtful cases as serious. Alcohol in breath is of no value.

Prognosis.—Serious symptom are:—

DURING ATTACK.—(1) Coma deep, lasting more than 24 hours; increasing depth suggests ventricular hæmorrhage. (2) Rapid rise of temperature within 48 hours. (3) Conjugate deviation persisting. (4) Respiration irregular or Cheyne-Stokes.

Cerebral Hæmorrhage—Prognosis, *continued.*

SUBSEQUENT TO ATTACK.—(1) Temperature persisting: should fall on 3rd or 4th day. (2) Acute bedsores. (3) Congestion of lungs. (4) Albuminuria. (5) Bilateral paralysis.

PARALYSIS.—No improvement if not commencing within three months. From cortical hæmorrhage, recovery may be complete; internal capsule, always some permanent paralysis. Contractures are permanent.

MENTAL CONDITION.—Mental powers rarely recovered completely: often irritable.

Treatment.—*See* p. 872.

II. CEREBRAL EMBOLISM AND THROMBOSIS.

(*Cerebral Softening*)

Etiology.—

EMBOLISM.—Origin usually from the heart, fragments arising from: (1) *A diseased valve*, usually mitral; in recurrent or infective endocarditis. Rare in first attack of rheumatic fever or chorea. (2) *An auricular clot*, commonly mitral stenosis, occasionally in puerperium. Rare: (3) Clot from aneurysm; (4) Patch of atheroma; (5) Pulmonary sepsis. (6) Sepsis elsewhere (very rare).

SITE.—Left middle cerebral artery most common.

SEX.—Women commoner, from frequency of heart disease.

AGE.—Mainly young adults.

SEPTIC EMBOLI.—Occur in: (*a*) Infective endocarditis; (*b*) Pulmonary sepsis; (*c*) Rarely sepsis in other parts, e.g., pelvis. (Possibly consist of a few micro organisms.)

THROMBOSIS.—Causes (aiding coagulation of blood): (1) *Arterial degeneration*, due to (i) arteriosclerosis, (ii) syphilitic endarteritis: lumen narrowed. (2) Blood changes and feeble circulation. Debilitating conditions. Also: (3) Around embolus. (4) Aneurysms. (5) Pressure on vessels by neoplasms.

SITE.—Middle cerebral artery most common.

Pathology.—Characteristic result: degeneration and 'softening' in area deprived of blood. *Initial change* in area affected is *anæmia*. If circulation not re-established by collaterals, subsequent changes are: (1) *Infarction*, anæmic or hæmorrhagic. (2) *Softening*: area affected becomes moist and softens from infiltration with serum, nerve fibres degenerate, neuroglia swells. (3) *Slow removal* of degenerated tissue, proliferation of connective tissue, and *scar formation*; occasionally a cyst forms. *Abscess* forms if embolus infective. *Inflammation* occurs around area involved.

Red, yellow, or white softening depends on amount of blood in area. Red and yellow mainly in the cortex, white in the white matter.

ANASTOMOSIS OF CEREBRAL ARTERIES, AND RESULT OF BLOCKING.—

Central branches, e.g., through anterior perforated space, are pure end arteries, whence softening in internal capsule and corpus striatum.

Cortical vessels.—Establishment of collateral circulation varies; is greater than injection experiments suggest. Branches of middle cerebral are chiefly end arteries, whence focalizing lesions. Collateral circulation greater when main stems blocked, especially in posterior cerebral.

Symptoms.—May be none, especially when 'silent areas' affected, or in elderly persons. In general, resemble cerebral apoplexy in onset. subsequent hemiplegia, transient or permanent. Distinguishing factors:—

EMBOLISM.—(1) Age: young adults. (2) Heart disease common. (3) Onset sudden: no premonitory symptoms. (4) Loss of consciousness rarely deep. (5) Convulsions common (cortex affected). (6) Emboli may also be in retina or other sites.

THROMBOSIS.—(1) Age: syphilis in adults; arteriosclerosis after middle age. (2) Premonitory symptoms common. (3) Onset gradual: paralysis may start in one hand and extend. (4) Loss of consciousness varies with extent of thrombosis: in syphilis usually absent. (5) Convulsions not common at onset.

Previous symptoms or prodromata may exist for weeks, from vascular disease: headache (especially in syphilis), giddiness, tinglings, deficient memory, difficulties in speech.

LOCALIZING SYMPTOMS.—(Commonest site, middle cerebral; next, posterior cerebral and vertebral. Others rare.)

MIDDLE CEREBRAL ARTERY.—(1) Main stem or perforating branches: permanent hemiplegia (internal capsule). (2) Main stem distal to perforating branches: aphasia and hemiplegia, often transient. Branches: (a) Inferior frontal: motor aphasia. (b) Ascending frontal and parietal: complete hemiplegia. (c) Temporo-parietal: word-blindness and right hemianopia. (d) Temporal: word-deafness. (Symptoms of aphasia apply to lesions on left side.)

POSTERIOR CEREBRAL ARTERY.—Homonymous hemianopia and, may be, hemianæsthesia (posterior part of internal capsule). Collateral circulation often fair.

ANTERIOR CEREBRAL ARTERY.—Progressive dementia or nil. Rarely affected.

VERTEBRAL ARTERY.—Usually left. Supplies bulb, but results often partial, or transient, owing to anterior spinal artery. *Acute bulbar paralysis and some hemiplegia.* Lesion often includes:—

BASILAR ARTERY.—Bilateral hemiplegia and bulbar paralysis with pyrexia, as in pontine hæmorrhage. Usually rapid death in coma.

INTERNAL CAROTID.—Variable symptoms, depending on freedom of anastomosis: often none; may be hemiplegia, transient or permanent. Thrombosis may spread into branches, whence hemiplegia and coma, usually fatal.

POSTERIOR INFERIOR CEREBELLAR ARTERY.—*See* DISEASES OF THE CEREBELLUM, p. 876.

Combined lesions occur, e.g., both posterior cerebrals, or one with opposite middle cerebral: *apraxia* often marked.

Cerebral Embolism and Thrombosis, *continued.*

Prognosis.—

A. DURING ATTACK.—

THROMBOSIS.—Serious if previous attacks, extensive disease of vessels, or in prolonged coma. Varies with site: recovery rare if basilar, internal carotid, or both middle cerebrals thrombosed.

EMBOLISM AND SYPHILITIC THROMBOSIS.—Rarely fatal, unless basilar affected. In these also second attacks rare (if syphilis treated); not uncommon in other forms of thrombosis.

B. PARALYSIS.—Unless improvement commences in two or three weeks, recovery is exceptional. Prognosis worse in thrombosis than embolism, owing to vascular disease.

Note.—Extent of paralysis at onset is not always the maximum, as the extent may increase with advance of thrombosis.

In general: prognosis for life good, but for recovery from paralysis poor.

TREATMENT OF CEREBRAL HÆMORRHAGE AND SOFTENING.

All movements to be avoided. Should not be roused from coma. Avoid active measures while diagnosis uncertain.

HÆMORRHAGE.—

In acute stage.—(1) Place in bed at *absolute rest.* Head somewhat raised. Neck free and not bent. *Turn on side if respiration impeded.* Wipe out mouth frequently. (2) Hot bottles to feet. Ice-bag or cold to head. (3) *Purge freely.* Croton oil ♏ii in butter on back of tongue; calomel gr. v or elaterium gr. ½; (4) *No alcohol* or stimulants. Food unnecessary. Fluids if coma prolonged. (5) *Venesection.* Indications: full tense pulse, cyanosis and distended cervical veins, stertorous respiration. Contra-indications: small weak pulse. Method: external jugular vein preferable, remove 8 to 10 ounces once only.

Trephine for meningeal hæmorrhage, remove clot, and ligature vessel or plug with sterile wax.

After acute stage.—Rest in bed two to four weeks. Avoid bedsores: keep skin clean and dry, prevent burns from hot bottle. Light diet. No alcohol, digitalis, or drugs (except placebo or for intercurrent affection).

EMBOLISM AND THROMBOSIS.—

(1) Place in bed with foot slightly raised. Keep warm.

(2) *Stimulants* if heart feeble: brandy, ammonia, ether, or digitalis.

(3) Contra-indicated are: venesection, free purging.

Amyl nitrite recommended by some authorities.

If *syphilitic*: treatment at once. Commence with mercury injections or inunctions.

PARALYSIS.—Wrap limbs in cotton-wool. Light massage after ten days. Electricity after two to four weeks, especially faradic current to muscles antagonistic to contractures. Encourage use of recovering muscles. Treatment useless after three months and with contractures present.

III. THROMBOSIS OF THE CEREBRAL SINUSES.

Primary Simple Thrombosis.—Rare.

ETIOLOGY.—(1) Weakly infants, especially with diarrhœa; or in old people: 'marantic thrombosis'. (2) Anæmia and chlorosis: rare: 'autochthonous sinus thrombosis'. Usually in superior longitudinal sinus.

SYMPTOMS.—Mental dullness, headache, vomiting or convulsions. May be thrombosis elsewhere, e.g. legs. 'Marantic thrombosis' often latent, found at autopsy.

Secondary Thrombosis.—Not uncommon. Due to extension of inflammation from structures near: usually septic.

CAUSES.—(1) *Middle-ear disease*, commonest cause. Usually chronic disease. More often through posterior wall of middle ear than from mastoid cells. (2) *Tuberculous caries of temporal bone.* (3) *Suppuration outside skull*, rare: erysipelas, carbuncle, disease in nose, throat, or orbit. Occasional causes: fractures, compression by tumours.

SITE.—*Lateral sinus* most common, from otitis media. Cavernous sinus, etc.

SYMPTOMS.—*Septicæmia with local symptoms.*

ONSET.—Usually sudden: pyrexia, rigors, sweats. Headache; often drowsy.

LATERAL SINUS.—Tenderness and œdema behind ear and in neck. Internal jugular vein may be involved: palpable as hard cord, pain on using neck muscles. If condition progresses, pneumonia, pulmonary sepsis, general pyæmia, and death.

CAVERNOUS SINUS.—Œdema of eyelids, exophthalmos, may be retinal hæmorrhages. Ocular nerves and 1st division of 5th nerve may be affected in wall of sinus, with resulting paralyses, corneal ulceration, and occasionally optic neuritis.

TREATMENT.—*Lateral sinus*: operation and evacuation of contents. Prognosis improving with early treatment, but always grave. *Cavernous sinus*, inoperable.

IV. ANEURYSM OF THE CEREBRAL ARTERIES.

Aneurysm of larger arteries uncommon. (Miliary aneurysms are not referred to in this section. For these, *see* p. 865.)

Etiology.—

AGE.—Usually middle age.

SEX.—Commoner in males.

CAUSES.—(1) *Emboli*, usually infective endocarditis; (2) *Endarteritis*, usually syphilitic. Vessel walls thus weakened.

SITE OF ANEURYSM.—(1) *Middle cerebral*: usually embolic.

Aneurysm of the Cerebral Arteries—Etiology, *continued.*

(2) *Basilar*: origin often doubtful. Less commonly: (3) Internal carotid. Others rare. *Size*: pea to walnut.

Symptoms (*see also* INTRACRANIAL TUMOURS).—Groups: (1) Those in which *rupture and apoplexy* constitute first symptoms. (2) Those with symptoms of *cerebral tumour*: often followed by rupture. Majority in these two groups. (3) Compression of cranial nerves, occasionally. (4) *Latent*: found at autopsy, death from other causes.

Intracranial murmur occasionally (more common from hæmic causes).

MIDDLE CEREBRAL ARTERY.—Usually in Sylvian fissure. Convulsions, hemiplegia.

BASILAR ARTERY.—(*a*) *Anterior portion*: compresses crus: 'crossed paralysis' of 3rd nerve and hemiplegia. (*b*) *Posterior portion*: compresses pons: hemiplegia often bilateral, various cranial nerves affected.

INTERNAL CAROTID.—Compresses optic nerve or chiasma (hemianopia), or 3rd nerve.

Diagnosis.—Rarely possible. Suspected with localizing symptoms and endocarditis or possibly syphilis.

Treatment.—When diagnosis definite, supplying artery may be ligatured (vertebral, internal carotid). General treatment of aneurysms.

CHAPTER CXXXII.

DISEASES OF THE CEREBELLUM.

Functions of the Cerebellum.—The principal functions of the cerebellum are the maintenance of equilibrium, muscle tone, and the co-ordination of muscle movements. The vermis is connected with both sides of the body. Each lateral lobe is connected with the same side of the body, and diseases thus produce effects on the same side as the lesion. The lateral lobe has an inhibitory action on the opposite cerebral hemisphere by tracts through the superior cerebellar peduncle.

Summary of Symptoms in Cerebellar Lesions.—Are on same side as lesion. (1) *Cerebellar ataxia*: (*a*) gait reeling and lurching, usually to side of lesion; (*b*) equilibrium disturbed; (*c*) dysdiadochokinesis; (*d*) inco-ordination. (2) *Paresis*. (3) *Atonia*: flaccidity of muscles. (4) *Vertigo*: feeling of rotation *away* from side of lesion. (5) *Nystagmus*: coarse, *towards* side of lesion. May be: (6) Tremors and choreiform movements. (7) Position of head: occiput towards shoulder, usually of affected side. (8) 'Skew deviation' of eyes. This combination of symptoms constitutes the 'cerebellar syndrome'.

Sensation, sphincters, mental condition unaffected. *Reflexes* variable, plantar reflex flexor.

I. TUMOURS OF THE CEREBELLUM.

Occur both in children and adults. Glioma, tubercle, endothelioma commonest. (*See* INTRACRANIAL TUMOURS.)

Symptoms.—(*A*) *General*; (*B*) *Cerebellar*; (*C*) *Pressure effects by extension.* In chronic tumours, other portions of the brain may take over the cerebellar functions, and the special symptoms be slight or absent.

A. GENERAL SYMPTOMS.—Early and severe; headache (often occipital), vomiting, and optic neuritis.

B. SPECIAL CEREBELLAR SYMPTOMS.—

VERTIGO very frequent: sensation that objects are revolving round the body, or that body itself is revolving: the feeling of rotation is *away from side of tumour* (cf. cerebellopontine tumours). Probably from affection of vestibular nerve paths, and thus a direct cerebellar symptom.

MOTOR SYMPTOMS.—

1. *Ataxia* (cerebellar ataxia)—Certain special features:—
   - *a.* Gait reeling and lurching (as on a ship's deck on a rough day). If lesion unilateral, usually but not invariably bears to that side; may attempt to compensate this by rotating body to other side.
   - *b.* Unaffected by opening or closing eyes (no Romberg's sign).
   - *c.* Dysdiadochokinesis: repeated movements more slowly and clumsily performed on affected side (e.g., rapid repeated supination and pronation of wrist).
   - *d.* Continuation of movements: with pronation and supination as above, on attempting to cease, movement continues temporarily on affected side.
   - *e.* Ataxia affects coarse movements (cf. cerebral cortical ataxia, which affects finer movements, e.g., buttoning).
2. *Paresis, asthenia*—Weakness of limbs on side of lesion, due to deficient cerebellar function and not pressure on pyramidal tracts (no spasticity).
3. *Atonia* on side of lesion. Muscles markedly flaccid.

Other less definite motor symptoms:—

Swaying common when standing. Tendency to fall backwards in vermis lesions.

Position of head.—In unilateral lesions, occiput may incline towards same shoulder.

Tremors, spasms, and rhythmical movements of head, trunk, and limbs may be present in cerebellar lesions, but are rare in tumours (Stewart and Holmes).

Tetaniform spasms occur but are very rare (Hughlings Jackson's "cerebellar fits").

OCULAR SYMPTOMS.—

1. *Nystagmus.*—Commonly present. Movements coarse on looking towards lesion, but may be fine in opposite direction: coarse in bilateral lesions.

Diseases of the Cerebellum—Symptoms, *continued.*

2. '*Skew deviation*' of eyes occasional. On affected side, in and downwards; on sound side, out and upwards. Also 6th nerve often compressed, i.e., weakness of external rectus.

SENSATION.—Never affected.
SPHINCTERS.—Unaffected.
REFLEXES.—Variable, may be increased or diminished. Plantar reflex is always flexor (pressure on pons may cause extensor response).

C. PRESSURE EFFECTS BY EXTENSION.—Not usually marked.

CRANIAL NERVES.—Rare except 6th (common).
PONS.—Spastic hemiplegia on opposite side, from pressure on pyramidal tracts.
'FORCED ROTATORY MOVEMENTS.'—Occasionally when 5th nerve affected (through middle cerebellar peduncle), body tends to rotate, usually *away from lesion.*

Diagnosis from tumours of cerebellopontine angle, and general diagnosis.—*See* INTRACRANIAL TUMOURS.

Prognosis and Treatment.—*See* INTRACRANIAL TUMOURS.

II. VASCULAR LESIONS OF THE CEREBELLUM.

Thrombosis of Posterior Inferior Cerebellar Artery.—Produces complex but characteristic group of symptoms, due to distribution to portion of cerebellum and medulla.

ONSET.—Rapid, without loss of consciousness.

ON SIDE OF LESION.—(1) Ataxia of limbs. (2) Anæsthesia of face and pharynx (descending root of 5th nerve). (3) Paralysis of palate and vocal cords affecting speech (nucleus ambiguus and vago-glossopharyngeal nucleus). May be: (4) Nystagmus to side of lesion; (5) Loss of taste.

ON OPPOSITE SIDE.—Anæsthesia of trunk, limbs, and sometimes face: to pain, heat, and cold, while light touch and muscular sense often escape (dissociated anæsthesia).

Occasionally: sympathetic nerve disturbance on side of lesion, viz., pupil small, palpebral fissure narrowed; tachycardia

Transient affection of 6th, 7th, and 8th nerves on side of lesion may occur: tinnitus and Ménière's symptom-complex, etc.

Cerebellar Hæmorrhage.—Rare. Symptoms indefinite. Usually superior cerebellar artery. Pain at back of head, repeated vomiting, followed by unconsciousness. May be rotation to side of lesion, skew deviation of eyes. If recovery, cerebellar syndrome present. Often fatal from rupture into 4th ventricle.

III. PRIMARY DEGENERATIONS OF THE CEREBELLUM.

A group of rare diseases.

Primary Progressive Degeneration—A familial disease.

Onset: age about 30 to 40 years. Progresses to death. *Symptoms* of cerebellar syndrome: most marked are: (1) Reeling gait and disturbance of equilibrium. (2) Tremors of head and limbs, and inco-ordinate movements. (3) Articulation: hesitating, scanning or explosive. (4) Nystagmus or irregular nystagmoid movements. Sensation, sphincters, pupils, and eye movements normal. No mental impairment.

Morbid Anatomy.—Primary degeneration of cerebellar cortex, with atrophy of cells of Purkinje and fibres to central nuclei of cerebellum. Afferent and efferent cerebellar tracts unaffected.

Olivo-ponto-cerebellar Atrophy (Thomas).—No familial or hereditary factors *Onset* in late life. Progresses slowly to death. *Symptoms* of cerebellar syndrome: most marked are: (1) Reeling gait and disturbance of equilibrium. (2) Tremors of limbs. (3) Articulation slow and scanning. May be nystagmus.

Morbid Anatomy (Thomas).—Atrophy of cerebellar cortex, bulbar olives, and gray matter of pons. Total degeneration of middle cerebellar peduncles. Partial degeneration of restiform bodies. Central nuclei of cerebellum but slightly affected.

Other varieties differentiated, all rare, include: sporadic forms resembling primary progressive degeneration, and due to interstitial changes; acute cerebellar palsies in children, with or without encephalitis (*see* PALSIES OF CHILDREN, p. 860); spino-cerebellar ataxia, closely allied to Friedreich's ataxia (*see* p. 796).

CHAPTER CXXXIII.

DISEASES OF THE MENINGES.

I. PACHYMENINGITIS.

(*Disease of the Dura Mater.*)

Varieties.—May be either of the outer or inner layer, respectively pachymeningitis externa and interna.

PACHYMENINGITIS EXTERNA.—

CEREBRAL.—Results from: (1) Fracture of skull and subsequent hæmorrhage. (2) Inflammation—rare: by extension from neighbouring tissues, e.g., syphilitic caries, middle-ear disease. *Symptoms*: indefinite; of compression, or masked by causal condition.

SPINAL.—(1) Chronic: not uncommon, from tuberculous caries of bone. (2) Acute: rare, from aneurysm, syphilitic caries, tumours. *Symptoms*: from implication of roots and pressure on cord.

PACHYMENINGITIS INTERNA.—(1) Purulent: by extension from pia; very rare. (2) *Hæmorrhagic*: may be: (a) Cerebral, viz., pachymeningitis (interna) hæmorrhagica; (b) Spinal, usually

Pachymeningitis—Varieties, *continued.*

mainly in cervical region, viz., pachymeningitis cervicalis hypertrophica.

Hæmorrhagic Pachymeningitis (*Hæmatoma of the Dura Mater*).—

CEREBRAL FORM.—Very rare except in old people with dementia of various types, e.g., dementia paralytica: very rarely in cachexia, or severe anæmia at other ages. All conditions are associated with wasting of convolutions.

MORBID ANATOMY.—May be: (1) Thin subdural membrane; (2) Subdural hæmorrhage; or (3) Both. Virchow considered initial lesion inflammatory, the membrane forming first and the hæmorrhage being secondary. Authorities not yet unanimous; some still believe the membrane to be result of clotting of hæmorrhage.

SYMPTOMS.—Absent or indefinite, e.g., headache, delirium, stupor, convulsions, etc.

SPINAL FORM.—Rarer than above: may co-exist with it and symptoms be masked, or symptoms as in type following.

Hypertrophic Pachymeningitis of the Cord.—Usually in cervical region (*pachymeningitis cervicalis hypertrophica*). Rarely in lumbar zone. This special type is probably a fibrosis and not hæmorrhagic.

ETIOLOGY.—Syphilis in some cases. Often no factor.

MORBID ANATOMY.—Great thickening of the dura mater, embedding and compressing nerve roots and cord. May involve one or more segments.

SYMPTOMS.—Due to involvement of anterior and posterior roots and compression of cord. (1) *Root pains* intense and bilateral: mainly arms and neck. Areas of hyperæsthesia and anæsthesia. Followed after few months by: (2) *Wasting and atrophy* in upper limbs, commencing in hand, with contractures and *'claw-hand'*. (3) *Spastic paraplegia* in lower limbs. Disturbance of sensation and sphincters.

COURSE.—Chronic: a few years.

DIAGNOSIS from: (1) Tumours of meninges: onset in tumours unilateral, progress more rapid; in later stages symptoms identical. (2) Syringomyelia: by absence of the special sensory changes. (3) Caries: tubercle very rarely produces similar symptoms without obvious disease of bone; root symptoms less marked. (4) Amyotrophic lateral sclerosis: by sensory changes and severity of pains in pachymeningitis.

TREATMENT.—Antisyphilitic or palliative.

II. MENINGITIS.

Leptomeningitis, disease of the pia mater, commonly referred to as 'meningitis', occurs in various clinical, bacteriological, and etiological types:—

1. Tuberculous meningitis.
2. Cerebrospinal meningitis.
3. Suppurative or septic meningitis.

4. Pneumococcal meningitis.
5. In various acute infections and specific fevers: rare. Most common: enteric, influenza. Occasionally: gonorrhœa, scarlet fever, mumps, etc.
6. Syphilitic meningitis. (Course chronic or subacute.)
7. Terminal infection in debilitating diseases: cancer, chronic nephritis, etc.

Less:—

8. Meningism.
9. Quincke's serous meningitis.

A summary of the general symptoms of meningitis is given under suppurative meningitis (*infra*). For tuberculous, acute cerebrospinal, and syphilitic meningitis, see the respective sections.

Suppurative or Septic Meningitis.—Secondary to: (*a*) Local disease, e.g., middle-ear disease, cerebral abscess, disease of cranial bones; (*b*) General or distant infections, e.g., general septicæmia, acute osteomyelitis.

MORBID ANATOMY.—Thick greenish exudation either at vertex or base, and often extending into cord, or may be maximum at point of origin. Brain tissue hyperæmic. Ventricles usually distended.

SYMPTOMS.—The chief symptoms of meningitis are:—

1. HEADACHE.—Severe and rarely absent.
2. VOMITING.—Of cerebral type (frequent; no retching or pain; independent of food).
3. PYREXIA.
4. PULSE.—Slow and irregular.
5. RESPIRATION.—Slow and irregular.
6. PUPILS.—Frequently unequal. Early stages, contracted; later, dilated.
7. STRABISMUS.
8. OPTIC NEURITIS.—Commonest in basal meningitis, but often absent.
9. CONSTIPATION.

Various.—*Rigidity of neck*, if cord involved. Cranial nerve affections in basal meningitis. Spasms of muscles when cortex irritated. Kernig's and Brudzinski's signs. Reflexes may be increased early, later absent: may be extensor plantar reflex (Babinski's sign).

Blood.—Leucocytosis often marked: may be absent.

Cerebrospinal fluid.—Under pressure. Protein present. May be cloudy. Polynuclear cells numerous. May be organisms present in films or on culture.

Later stages:—

RESTLESSNESS.—Irritability. Teeth-grinding common.
PULSE.—Rapid and feeble. Temperature variable.
RESPIRATION.—Often Cheyne-Stokes type.
DELIRIUM.—Passing into terminal *coma*.

DURATION.—A few days.

Pneumococcic Meningitis.—

ETIOLOGY.—(*a*) Primary: either alone or with pneumococcic

Pneumococcic Meningitis, *continued.*

septicæmia. (c) Secondary: (a) local disease, e.g., otitis media, (b) distant infections, e.g., empyema, pericarditis.

MORBID ANATOMY.—Exudation markedly thick and profuse, either at vertex or base. Cord rarely escapes.

SYMPTOMS.—*See* SUPPURATIVE MENINGITIS. Onset usually very rapid, and duration short (1 to 3 days). Invariably fatal.

Note.—'Meningism' is common in acute pneumonia.

Enteric Fever; Influenza.—Symptoms of meningitis may be due to: (a) Meningism, commonly. (b) True meningitis, very rarely; either (i) specific organism, or (ii) pyogenic organism. *Recovery may occur.*

Meningism.—During acute specific fevers a condition may occur in which symptoms resemble, or are identical with, meningitis, but in which recovery occurs, or at autopsy no changes of meningitis are present. Especially common in enteric fever, also in acute pneumonia: occasionally occurs in middle-ear disease, and alcoholism.

DIAGNOSIS FROM MENINGITIS.—Suggestive of meningism are: (1) Onset early in disease; (2) Onset rapid; (3) Slow pulse and respiration less frequent; (4) Kernig's sign usually absent; (5) Cranial nerves rarely affected, except strabismus; (6) Cerebro-spinal fluid: no changes of meningitis, but may be increased pressure.

CHAPTER CXXXIV.

GENERAL DISEASES WITHOUT RECOGNIZED ANATOMICAL BASIS.

I. PARALYSIS AGITANS.

(*Parkinson's Disease. Shaking Palsy.*)

A chronic disease of later life, characterized by peculiar tremors, rigidity, attitude, expression, and gait. Not uncommon.

Etiology.—

AGE.—Usually 50 to 60 years. Rare under 40.

SEX.—Males twice as common as females

HEREDITY.—Instances rare.

EXCITING CAUSES.—Ascribed to mental worries, or exposure to wet or cold. Occasionally sudden onset following such stimuli. Syphilis and alcohol: no influence. Closely similar condition, often developing rapidly, may occur in encephalitis lethargica.

Morbid Anatomy.—No constant changes. In nervous system, especially cord, may be thickening of small vessels and overgrowth of neuroglia connective tissue, as in senility, but not invariable. Clinically, points to changes in cerebral cortex. S. A. Kinnier Wilson suggests corpus striatum, from certain resemblances to progressive lenticular degeneration.

Symptoms.—

ONSET.—Gradual. Very rarely, rapid. *Tremors* usual initial symptom; commence in one hand, then same leg (unilateral paralysis agitans), later on other side, and general symptoms. Aching and stiffness may precede tremor.

STAGE OF INVASION.—1 to 3 years.

CLINICAL CONDITION FULLY DEVELOPED.—Characteristics: (1) *Tremors*; (2) *Rigidity*; (3) *Attitude*; (4) *Facies*; (5) *Gait*.

TREMORS.—Typically in hands. General character of movements: Regular and rhythmical; at first fine, rate 5 to 7 per second; later coarser and slower. *Increased by rest* and emotion. Checked temporarily by will or voluntary movement. Cease in sleep.

Hands: 'Pill-rolling' movements of fingers, with pronation and supination of forearm, occasionally some flexion and extension of wrist. *Position of fingers*: metacarpophalangeal joints flexed with phalangeal joints extended ('interosseal position') or flexed: thumb opposed to index finger. Large joints of arm rarely affected.

Legs: Ankle-joints most commonly affected.

Head: Not often affected; occasionally to-and-fro movements. Face very rare: eyes never.

RIGIDITY.—Progressive: cause of attitude, expression and gait.

Muscular weakness progressive, but no absolute paralysis

Voluntary movements all slow and deliberate.

ATTITUDE.—Characteristic. Stands with head bent forward, back curved and rigid; arms flexed at elbows, held away from body, hands in front of abdomen.

FACIES.—'Parkinson's mask'. Expressionless and changeless. Eyebrows often elevated, forehead smooth or wrinkled.

GAIT.—A hurried shuffle, "running after the centre of gravity" ('*festinant*' or propulsion gait). Starts slowly, and has difficulty in stopping. If pulled backwards, makes several rapid steps and tends to fall ('retropulsion'). Attitude, as described, continues on walking and is cause of gait.

Gait may be normal for several years after tremors.

VOICE.—Often shrill and monotonous. Hesitation followed by rapid speech.

SENSORY DISTURBANCES.—(1) Sensation of great heat common. May be sweating and local rise of temperature. (2) Cramps and aches common. Often severe in later stages, causing restlessness. Cutaneous sensation normal.

SKIN.—Sometimes thickened.

MENTAL CONDITION.—Unchanged.

Sphincters unaffected. *Reflexes* normal, or increased.

Course and Prognosis.—Incurable. Gradual advance, with periods of improvement. Becomes bedridden. Death from pneumonia or other intercurrent disease.

DURATION.—8 to 19 years. Rarely 20 to 30 years.

Paralysis Agitans, *continued.*

Diagnosis.—Usually at sight. Difficulty in atypical cases, or in absence of a characteristic symptom, e.g.: (1) *Tremors absent*, other signs often well marked; (2) Tremor alone; (3) Unilateral distribution. Generally these cases are in an early stage, and completer syndrome develops later. Diagnosis also from:—

SENILE TREMORS.—Face muscles early affected.

OSTEO-ARTHRITIS.—Thickening or grating of joints.

Treatment.—Does not cure, but alleviates suffering *Indications*: to maintain strength and diminish tremors and rigidity.

GENERAL.—Quiet life. Massage, active and passive movements, warm baths: inhibit rigidity and aid nutrition. In later stages, comfortable bed and light bed-clothing.

DRUGS.—Hyoscine hydrobromide. Either: (1) Hypodermically, gr. $\frac{1}{100}$ to $\frac{1}{80}$ at night; or (2) By mouth, gr. $\frac{1}{150}$ increasing to gr. $\frac{1}{80}$ night and morning. Considerable relief. Beware of toxic action. For sleeplessness, barbitone. For aching pains, aspirin or salicylates.

II. CHOREA.

(*Sydenham's Chorea. St. Vitus' Dance.*)

A disease mainly of childhood, characterized by irregular involuntary muscular spasms, and by frequent occurrence of endocarditis. It is closely connected with acute rheumatism.

Etiology.—

AGE.—Usually between 5 and 15 years. Rare under 5 and over 20 years, except in pregnancy.

SEX.—Females form 70 per cent. Over 20 years nearly all females.

INHERITANCE.—(*a*) Rheumatic family history 15 to 20 per cent; (*b*) Nervous family common. Red hair frequent in chorea.

RELATION TO ACUTE RHEUMATISM.—Close relationship of chorea shown by:—

1. Frequency of acute rheumatic arthritis either (*a*) previously, or (*b*) immediately before chorea.
2. Frequency of acute endocarditis; also of pericarditis
3. Other symptoms occurring in both, e.g., tonsillitis, subcutaneous nodules.

RELATION TO MENTAL DISTURBANCE.—Close. Subjects usually bright, clever, excitable children. *Sudden strain*, e.g., fright: chorea may be immediate or after few days. *Chronic strain*: overwork at school of special importance.

RELATION TO PREGNANCY.—Not infrequent, especially if emotion great. Characteristics: (1) First pregnancy most common. (2) Onset about 3rd month. (3) Often severe, may be maniacal; considerable mortality. (4) May recur in successive pregnancies. (5) Rarely, after abortion or full time. (6) Frequency in illegitimate pregnancies not proved.

OTHER ETIOLOGICAL FACTORS OF LESS IMPORTANCE.—*Acute infectious fevers.*—No relation except to scarlet fever with arthritis.

Imitation.—Never a cause.
Hysteria.—May simulate chorea; and is origin of 'epidemics of chorea'.
Reflexes.—Irritation by worms, adenoids, ocular defects: no relation beyond effect on health.

Morbid Anatomy.—No constant lesions. Changes in nerve-cells multiple minute areas of softening, and embolism of small vessels in the brain. Acute endocarditis in 90 per cent of fatal cases.

PATHOGENESIS.—Two predominating factors, often co-existent: (1) Acute rheumatic manifestations; (2) Mental overstrain, acute, chronic, or inherited.

Seat of lesion in cerebral cortex suggested by: (a) Spasmodic movements, i.e., affection of motor centres; (b) Cessation in sleep; (c) Paresis; (d) Hemichorea. Possibly also cerebellar cortex, suggested by (a) Hypotonus; (b) Inco-ordination.

Poynton, Holmes, and Paine described small cortical lesions with presence of rheumatic diplococci.

Kirkes' theory.—Multiple minute cerebral emboli from endocarditis. Unproved.

Symptoms.—

GENERAL DESCRIPTION OF MOVEMENTS.—Irregular, involuntary, purposeless, spasmodic movements.

IRREGULAR.—Same movement is not repeated, as it is in tics.

INVOLUNTARY, but, with mental effort, movements can be inhibited temporarily and a voluntary movement performed.

PURPOSELESS, but muscles contract in sequence as in performing a voluntary movement; differing from the contraction of a single muscle, e.g., platysma myoides, as in certain nervous conditions.

SPASMODIC.—Movements sudden and of short duration.

DURING SLEEP.—Movements usually cease.

All possible movements, voluntary and of expression, may occur, and in all grades of severity.

Other factors are: (1) *Paresis, some degree invariable*; (2) Inco-ordination; (3) Hypotonus. Assessment and separation of these factors in given case usually impossible.

DISTRIBUTION OF MOVEMENTS.—Frequency: (1) Hands or face; (2) Legs; (3) General. *Hemichorea* common, especially right, but bilateral in face. Slight movements well exhibited on extending arms with fingers widespread and simultaneous protrusion of tongue.

MODES OF ONSET.—(1) Movements rapidly develop. (2) Dropping of articles. (3) Dragging one leg. (4) Changes in temperament; dullness or irritability.

CLINICAL CONDITION DEVELOPED.—

MOVEMENTS.—As described. Facial expressions, eyebrows, tongue, jerks of head; movements of fingers, hands, shoulders, upper extremities; legs; trunk. *Respiration*: often affected, e.g., sudden spasmodic inspiration. *Mastication and deglutition*: difficult in severe cases.

Chorea—Symptoms, *continued.*

SPEECH.—Often impaired, jerky. *Aphasia*: occasionally complete for weeks in severe cases: never permanent.

CARDIAC SYMPTOMS AND DISEASE.—Rarely cardiac pain or complaints. Heart rapid. Apex beat often diffuse. Hæmic murmurs (base or less often apex) not uncommon. No dilatation, or displacement of apex beat

ORGANIC HEART DISEASE.—Very frequent. *Note*: (1) Present at onset from previous rheumatism (history often absent); or (2) Develops during chorea or afterwards: occurs in 50 per cent at least. (3) Becomes commoner with each recurrence. (4) *Acute endocarditis* usual form: generally mitral valve, stenosis frequent. Present in 90 per cent of fatal cases. Ulceration rare. Embolism rare. (5) *Pericarditis* common.

TEMPERATURE.—In severe cases rarely absent, but slight and of short duration. Continued pyrexia suggests endocarditis or arthritis. Hyperpyrexia, usually with pericarditis or delirium.

REFLEXES.—No constant change. *Knee-jerk*: response often delayed and then contraction 'sustained'. May be due to (*a*) increased reflex, or probably (*b*) a choreic movement or pseudo-reflex.

PARESIS.—Usually slight. Rarely, severe (movements often slight); flaccid type; never permanent ('paralytic chorea').

MENTAL DISTURBANCES.—Common but slight: dullness or irritability. Rarely mania; and then usually in adult females and pregnancy ('chorea insaniens')

SUBCUTANEOUS NODULES.—On fibrous structures: especially palpable at point of elbows and wrists; usually multiple, rarely larger than pea. *Presence serious*: pericarditis often occurs.

ANÆMIA develops in later stages.

Of less importance: *Sensory symptoms*; pain very rare. No changes in sensation. *Sphincters* unaffected. *Electrical reactions*: normal. *Pupils* usually dilated: hippus may occur *Skin*: occasionally various 'rheumatic' eruptions, e.g., erythemata, purpura. *Urine*: urea excretion high.

Varieties.—Certain special types: (1) Hemichorea; (2) Paralytic chorea; (3) Chorea of pregnancy; (4) Chorea insaniens; (5) Chorea gravis—movements of great severity.

Course and Prognosis—

DURATION.—Variable. Movements rarely exceed two months. Relapses common.

RECOVERY from immediate attack usually complete: when severe, slight movements may persist, increased on excitement.

RECURRENCES.—Frequent, especially in spring.

MORTALITY.—About 2 per cent.

TEST OF SEVERITY OF ATTACK.—Consider: (1) Extent of involuntary movements. (2) Extent of performance of voluntary

movements. (6) Affection of speech. (7) Dangerous symptoms and conditions, as below.

DANGEROUS SYMPTOMS AND CONDITIONS—

1. Acute endocarditis. Affects remote more than immediate prognosis. Embolism rare.
2. Pericarditis. Subcutaneous nodules often precede onset.
3. Hyperpyrexia. Pericarditis frequently present. Prognosis serious.
4. Chorea of pregnancy. Often severe.
5. Chorea insaniens, and severe psychical disturbance. Most frequent in last group.
6. Chorea gravis Exhaustion may be fatal.

Diagnosis.—Usually simple. Main difficulties :—

HYSTERIA.—Movements purposeful, usually repeated. Worse on order to control.

TICS AND HABIT SPASMS.—Repetition of similar movement.

In rare maniacal and paralytic choreas, movements occasionally overlooked.

Difficulties rarely occur with other tremors, e.g., athetosis, Friedreich's ataxia.

Treatment.—Two essentials : (1) Complete rest ; (2) Full diet.

REST.—Complete rest in bed, for body and brain, i.e., no books or games. At least four weeks, and until movements completely subside.

DIET.—Commence with milk (5 pints), cream, eggs, and bread and butter. Full diet in few days with extra milk and cream, or in milder cases from onset.

DRUGS.—Little value and action on spasms slight. In most use are :—

ASPIRIN gr. xl to lx, daily. Especially if rheumatism. Salicylate of sodium similarly, but less valuable

ARSENIC.—Good tonic : no proved effect on spasms. Fowler's solution, ♏ij, t.d.s. ; increase ♏j, alternate days, to ♏xv ; well diluted and after food. Watch for signs of excess, and if occurring remit for one week.

SEDATIVES.—Chloral and sod. bromide, āā gr. v to x, t.d.s., or chloretone. Diminish spasms, but *subsequent mental derangement not infrequent*, especially in severe cases, less often in milder types, but sedatives here unnecessary. Spasms often return on remitting drug. Hence use only in severe cases where other measures fail. Remit if cardiac weakness occurs. Bromide little value without chloral. Sedatives must not be employed as a routine treatment.

SEVERE TYPES.—Water bed. Wet packs (either cold or tepid). Stimulants. Sleep often prevented and is essential obtain rest by : (1) Chloral and bromide or chloretone; (2) Chloroform inhalations. Morphia rarely succeeds if chloral fails.

ACUTE ENDOCARDITIS.—*See* ENDOCARDITIS.

CONVALESCENCE.—Treatment of great importance.

FRESH AIR. *Moderate exercise.* Long night and rest on couch part of day.

Chorea—Treatment, *continued.*

FULL DIET. Tonics of iron and strychnine. Cod-liver oil or similar preparations.

CORRECT ALL SOURCES OF IRRITATION, viz., ocular defects, adenoids, etc.

WARN PARENTS AGAINST: possibility of recurrences, of heart disease, and of ill-effects of mental worry, *especially examinations.*

III. HUNTINGTON'S CHOREA.

(*Chronic Hereditary Chorea.*)

A rare disease characterized by: (1) Choreiform movements; (2) Onset in middle life; (3) Progressive mental weakness; (4) Usually hereditary and familial.

Etiology.—

AGE.—Onset at 30 to 40 years. Both sexes.

HEREDITY.—Has been traced through many generations: transmitted by both sexes. If one generation escapes, does not recur.

Morbid Anatomy.—Lesions in central nervous system, but not pathognomonic: general resemblance to dementia paralytica.

1. MEMBRANES.—Chronic pachymeningitis and inflammation of pia-arachnoid.
2. CHRONIC ENCEPHALITIS.—Atrophy of convolutions: primary parenchymatous degeneration of neurons. Probably the last is the essential change.

Symptoms.—

ONSET gradual: movements before mental changes

MOVEMENTS.—As in chorea, but slower, and *inco-ordination marked.* Commence in hands and face. In early stage controlled by will, and voluntary movement possible. Later severe and universal. Much facial contortion, speech difficult owing to tongue spasms, gait lurching.

MENTAL DISTURBANCE.—Attacks of depression or excitement: may be suicidal: progresses to complete dementia.

Course.—Progressive. Life often not shortened.

Treatment.—Palliative only. Arsenic and tonics.

SENILE CHOREA.

Onset usually after 50 years. Occasionally ascribed to anxiety or fright.

Movements as in chorea. No relation to rheumatism or endocarditis.

Morbid Anatomy.—Resembles Huntington's chorea. Regarded by many authorities as a sporadic form (probably correctly), and course may be identical, but differs in following: mental changes are rarer, recovery may occur, no hereditary or familial factors (brothers, sisters, and children unaffected).

CONGENITAL CHOREA.

Very rare Movements present from birth and persist No spasticity Mentally slow but not idiots Related to Huntington's chorea

Note – *Cerebral diplegias* with choreiform movements show definite spasticity

IV. MIGRAINE.

(*Hemicrania*)

A condition characterized by paroxysmal attacks of headache, usually with nausea, and often preceded by disorders of vision

Etiology. –

AGE – First attack usually between 5 and 20 years rarely after 30

HEREDITY —Common Gout and neuroses not infrequent in family Mental ability often above normal Rarely in outdoor occupations

EXCITING CAUSES —Mental worries gastric disturbances; ocular defects menstruation Often no evident cause

RELATION TO EPILEPSY —Rarely, attacks apparently alternate, but relation slight

Pathogenesis. — No histological changes Numerous theories include —

(1) SPASM OF ARTERIES —Supported by vasomotor phenomena, spasm of and occasionally sclerosis of temporal and retinal arteries and transient paralyses Against this is apparent absence of vasoconstrictor fibres in the cerebral arteries

(2) INSTABILITY OF CENTRAL NERVOUS SYSTEM Akin to epilepsy and supported by similarity of phenomena Various stimuli may excite attack

(3) Other theories suggest dietetic and metabolic errors and protein hypersensitiveness

Symptoms.—

CHARACTERS OF ATTACK —Premonitory symptoms common, especially visual, followed by headache and nausea

PREMONITORY SYMPTOMS —

1 VISUAL PHENOMENA —Very common Two forms, often combined (a) Alterations in vision Often 'steaminess' of sight, or small central blind spot gradually extending, may be homonymous hemianopia (b) Occurrence of colours usually very brilliant Often commence centrally and spread outwards in bands or 'fortification figures'. All degrees from specks to formed objects

2 SENSORY PHENOMENA —Unusual Tingling in an extremity, spreading slowly to head usually opposite side to headache Subsequently slight paresis, or sometimes anæsthesia

3 VERTIGO, SLIGHT INCOHERENCE, OR APHASIA —Occasionally.

Migraine—Symptoms, *continued.*

HEADACHE.—Follows premonitory symptoms.

ONSET.—Usually in one spot, temple, eyeball, etc. Extends and increases: usually unilateral, i.e., hemicrania; occasionally pain extends into neck, rarely arm, or both sides of head. Scalp tenderness common. Intensity varies: often extreme. Character boring or throbbing. Increased by movement, noise, light, or erect position.

NAUSEA.—Rarely absent. Anorexia marked. Vomiting occasionally: may be recurrent.

VASOMOTOR PHENOMENA.—Occasionally: sometimes marked. May be unilateral. Face and extremities pale and cold, pupils small; later hyperæmia. Pulse may be slow. Temporal arteries may be in spasm.

VARIATIONS IN ATTACKS.—Headache and nausea without premonitory symptoms common. Less often, marked visual phenomena with slight headache.

DURATION.—Usually one day, ends with night's rest; if severe, subsequent malaise one to two days. In rare cases, may be subsequently a transient aphasia, paresis, or complete blindness.

Course.—Attacks recur for years. Subject often aware of approach of an attack before definite premonitory symptoms. Often monthly or periodically, but frequency varies. In same individual, attacks may resemble each other closely, or may vary greatly. *Cessation* usual about age of 50, or after climacteric: sometimes with removal of exciting cause

Diagnosis.—

EPILEPSY.—In migraine: (1) Prolonged premonitory symptoms; (2) Visual phenomena; (3) No unconsciousness or spasms; (4) Severity of headache.

CEREBRAL TUMOUR.—In migraine: (1) Long duration, (2) Long intervals of freedom; (3) No optic neuritis; (4) Visual phenomena.

CHRONIC NEPHRITIS.—In migraine: no urinary changes.

Note.—After attack of migraine, much pale urine may be passed.

Treatment.—Subject sometimes learns, and occasionally averts, exciting cause.

GENERAL HYGIENE.—General healthy life, with outdoor exercise. Also:—

1. Treat any exciting cause, especially ocular and gastric defects.
2. Diet. Vegetarian diet, or reduction of meat, often, but not invariably benefits. Some subjects need meat. Alcohol best omitted.
3. Bowels. Strictly regulated.

PREVENTION OF ATTACKS.—Often defies treatment. Among drug methods, best are: (1) Bromides, long course, as in epilepsy, (2) Nitroglycerin. Tablets, gr. $\frac{1}{100}$, bis die, or liq. trinitrini, ½ to 1. Especially with high blood-pressure. Other drugs include: cannabis indica, belladonna, gelsemium.

TREATMENT OF ATTACK.—Rest in a quiet dark room. Warmth to feet. Hot drink. No alcohol. At earliest warning, a saline purge and calomel. *Local*: cold compresses to head. *Drugs* vary with subject: best are aspirin gr. x to xxx, or phenacetin gr. viij with caffeine gr. ij, or pyramidon gr. vij to x. (Most subjects prefer to be undisturbed.) Tonic after the attack.

V. NEURALGIA.

Paroxysmal pain in course of a nerve in absence of organic disease of the nervous system. This definition excludes neuritis. The pain and symptoms may be identical with, and the distinction difficult from, conditions with an organic basis. Visceral referred pains are not true neuralgia.

Etiology.—

AGE.—Usually middle life.

SEX.—Commoner in women.

PREDISPOSING CAUSES.—(1) *Neurotic taint*; (2) *Anæmia and debility* of all forms; (3) Influenza, enteric fever; (4) Gout, alcohol, lead poisoning, diabetes, malaria (this group is probably neuritis). May be good health.

EXCITING CAUSES.—*Cold*, constipation, worry, peripheral irritation.

Symptoms.—

PAIN.—General characters:—

PAROXYSMS every few seconds to minutes; burning or shooting. Between paroxysms: dull ache or painless.

DISTRIBUTION.—Unilateral. In course of a nerve, nerves, or division of nerves: but may spread widely in height of paroxysm.

'TENDER SPOTS'.—Tenderness mainly at certain spots in course of nerve: usually at emergence through fasciæ or bone.

RECURRENCES usual.

VASOMOTOR AND TROPHIC CHANGES. Occasional. Skin during paroxysm cold, and later hot (often feels numb). Rarely, erythema or œdema over area: when chronic, hairs may whiten and fall out.

Diagnosis.—

ORGANIC DISEASE of nervous system or viscera to be excluded.

NEURALGIA.—Usually: (1) Unilateral; (2) Intermittent; (3) Tenderness mainly at certain tender spots; (4) No muscular wasting; (5) No anæsthesia.

REFERRED PAIN OF VISCERAL DISEASE.—Pain and superficial tenderness in areas not of peripheral nerve distribution.

NEURITIS.—(1) Pain more continuous; (2) Whole course of nerve tender; (3) Muscular wasting. Diagnosis may need long observation.

Treatment.—

INITIAL.—Treat any peripheral irritation. Reassure patient of absence of organic disease.

Neuralgia—Treatment, *continued.*

LOCAL TREATMENT.—Mainly counter-irritation.

1. HEAT OR COLD.—Hot bottle or poultice.
2. SEDATIVE APPLICATIONS.—(1) Menthol. (2) Liniment of aconite, belladonna, and chloroform ('A.B C.'). (3) Freeze tender spots with ethyl chloride.
3. COUNTER-IRRITATION.—Mustard. Blisters (liq epispasticus). Cautery. Leeches. Electricity.
4. INJECTIONS OF ALCOHOL INTO NERVE TRUNK.

GENERAL TREATMENT.—Treatment of predisposing causes: tonics, cod-liver oil. Diet plain; meat in moderation only. Regulate bowels. Regular exercise. Massage. Change of air. Alcohol often effective, but needs care.

SPECIAL ANALGESIC DRUGS.—Tincture of gelsemium (♏ x, t d s.). Butyl chloral hydrate (gr. xxx to l, daily). These two particularly in head neuralgia. Note that butyl chloral is incompatible with alcohol: can be combined with gelsemine hydrochloride gr. $\frac{1}{20}$, t.d s. Pyramidon (gr. vii to x, t d s), phenacetin, aspirin. Any of these often effective. Also quinine. Morphia and cocaine to be avoided.

SPECIAL CLINICAL VARIETIES.

Neuralgia of Fifth Nerve and Allied Conditions.—*See* TRIGEMINAL NEURALGIA, p. 838.

Cervico-occipital Neuralgia.—

NERVES INVOLVED.—Posterior branches of cervical 1 to 4. Often bilateral.

PAIN.—Back of head and neck, especially along great occipital.

TENDER SPOTS.—Midway between spine and mastoid processes.

SCALP.—Extreme hyperæsthesia common

ETIOLOGY.—*Cold.* Also in cervical caries.

SPECIAL TREATMENT.—Division of nerves.

Brachial Neuralgia.—

NERVES INVOLVED.—Branches of brachial plexus.

PAIN.—Shoulder, axilla, along inner arm (ulnar nerve) to fingers.

TENDER SPOTS.—Behind elbow (ulnar). Axilla. Posterior border of deltoid (circumflex).

ETIOLOGY.—Ordinary causes, but injury common and cold rare. Increased by movement. Closely related to brachial neuritis and arthritis of shoulder-joint.

Intercostal Neuralgia.—

NERVES INVOLVED.—Anterior branches of dorsal nerves 2 to 9. Common.

PAIN.—Constant ache, with paroxysms. Increased by respiration.

TENDER SPOTS.—At three cutaneous branches, viz., near spine, mid-axilla, near sternum.

SUPERFICIAL TENDERNESS.—Often severe.

ETIOLOGY.—Common in: (1) Women, especially with hysteria; (2) Herpes zoster, before and after eruption.

DIAGNOSIS.—From (1) Spinal disease: tabes, caries, aneurysm, tumour. (2) Callus of fractured ribs. (3) Angina pectoris. Also (4) Acute lung conditions.
TREATMENT.—Counter-irritation, blisters. Prognosis good.

Mastodynia (*Neuralgia of Breast*).—
NERVES INVOLVED.—Intercostals supplying breast (2 to 6). Usually on left.
ETIOLOGY.—Women, middle-age. Debility, pregnancy, over-lactation.
SPECIAL TREATMENT.—Ascertain, and assure patient of, absence of neoplasm.

Lumbar Neuralgia.—
NERVES INVOLVED.—Lumbar plexus.
TENDER SPOTS AND PAIN.—Commonest: iliac crest, scrotum, pubium magnus. Occasionally: 'irritable testis', spermatic cord, inguinal canal.
LOCAL CAUSES.—Constipation, pelvic disease.
'CRURAL NEURALGIA.'—Mainly front of thigh (anterior crural).
Colon disease, sciatica.

Coccygodynia.—
NERVES INVOLVED.—Coccygeal plexus.
ETIOLOGY.—In women: hysteria, after labour, etc.
PAIN. Severe and obstinate. Increased by sitting.
SPECIAL TREATMENT.—Removal of coccyx: not always successful.

Metatarsalgia (*Morton's Disease*).—
PAIN.—In 4th metatarso-phalangeal articulation, may extend up leg. Unilateral.
ETIOLOGY.—Usually women. Morton's explanation: head of 5th metatarsal squeezed under 4th and pinch metatarsal nerve (doubtful).
DIAGNOSIS.—From acute rheumatism.
SPECIAL TREATMENT.—Avoid tight shoes; treat flat-foot. Finally, excision of head of 4th metatarsal.

Other Neuralgias of Feet.—Often from flat-foot.
PAINFUL HEEL.—Often, not invariably, gonorrhœa.
PLANTAR NEURALGIA (e.g., tender toes in enteric).—Often neuritis.

Visceral Neuralgia.—*See* GASTRIC NEUROSES, etc.; also HYSTERIA; NEURASTHENIA.

VI. OCCUPATION NEUROSES.

Inability to perform, and usually pain on attempting, some professional muscular action: following its frequent repetition over a considerable period, and without organic disease. Occupations affected are complex acts, carefully learnt, but by repetition becoming practically automatic. Disability applies solely to the special act, and the muscles can be used freely in other groupings and actions.

Occupation Neuroses, *continued.*

Nomenclature.—The term 'cramp' is applied to the various conditions, but spasm is often absent, and hence affection is usually considered a true neurosis, a disturbance of the nerve centres. 'Writer's cramp' is the typical and commonest form.

Writer's Cramp.—

ETIOLOGY.—Age 20 to 45 years. Both sexes liable, but males predominate.

PREDISPOSING CAUSES.—(1) *Overwork*, common; (2) *Faulty position* in writing. Occasionally (3) Slight injury; (4) Nervous disposition.

FAULTY OR STRAINED POSITIONS.—Specially affects those writing with wrist or little finger as fixed position. Correctly, the fingers should be used only to hold pen, and that lightly, and practically all movements made from wrist or forearm, forearm resting on table.

SYMPTOMS.—Commonest form is inability to keep index finger on pen. Complaints may be: (1) *Pain*. 'Neuralgic type'. (2) *Weakness*. 'Paralytic type'. (3) *Spasms*. 'Spastic type'; uncommon; spasm may throw pen from hand. Distinction of types unimportant: pain and weakness often inseparable.

ONSET.—Gradual. First at end of long day and in fingers only. Later, immediately on writing. If persisted in, spreads to forearm and even shoulder.

TENDERNESS over nerve trunks in severe cases. May be local œdema.

SENSATION AND ELECTRICAL REACTIONS normal. WASTING rare and slight.

COURSE AND PROGNOSIS.—Always increases if action continued. Prognosis best in mild forms, of short duration and after injury. Prognosis bad with: (1) Neurotic taint; (2) Long duration, (3) Faulty position. Long rest may cure, but relapse common.

DIAGNOSIS.—Many diseases disturb writing, and examination must be fully made for:—

1. ORGANIC DISEASE OF NERVOUS SYSTEM.—As dementia paralytica, hemiplegia, disseminated sclerosis, etc.; also paralysis agitans. Dystrophies, myelopathies, cervical rib and lesions of brachial plexus and its branches, syringomyelia.
2. LOCAL DISEASE.—As osteo-arthritis, tenosynovitis, neuritis.

Also examine: (1) For neuroses in patient and family. (2) Action of writing: of essential importance. 'Phobia' of writer's cramp not uncommon.

TREATMENT.—

REST from the action: immediate and complete. If mild, three months. Holiday preferable.

SEVERE CASES.—Rest nine months. Tonics. Massage and gentle exercises for hands (after pains subsided).

RE-EDUCATION of method of writing from commencement, i.e., copy-books. Operations useless.

IF RECURRENCE.—Write with left hand or use typewriter.

Other Varieties of Occupation Neuroses.—Numerous. General facts and treatment as in WRITER'S CRAMP

'PIANIST'S CRAMP'.—Not uncommon

'TELEGRAPHIST'S CRAMP'—Very rare

'TYPEWRITER'S CRAMP'—Excessively rare

VII. EPILEPSY.

A disorder of the nervous system characterized by repeated attacks of loss of consciousness, often associated with convulsions Two principal types (1) *Grand mal* or major epilepsy unconsciousness with convulsions (2) *Petit mal* or minor epilepsy unconsciousness without convulsions Also Jacksonian epilepsy, in cortical lesions convulsions without unconsciousness, this type and certain epileptiform convulsions are etiologically distinct from true epilepsy

Etiology.—

AGE—Onset commonest under 20 years 75 per cent before 20 years No age quite immune, but onset of true epilepsy rare after 30 years Onset common in infancy, puberty and second dentition

SEX—Equal in childhood In later decades males in excess Frequency of attacks increased at menstruation especially when irregular Catamenia and pregnancy, no influence

SPECIAL FACTORS—

1 HEREDITY—Direct inheritance not infrequent (statistics vary) Also epilepsy, insanity and neuroses common in family history, direct or collateral

2 ALCOHOL—Chronic alcoholism in parents in high percentage in certain statistics definite relation unproved Epileptiform convulsions occur in chronic alcoholism

3 INFANTILE CONVULSIONS—Precede epil , not infrequently

4 REFLEX IRRITATION—Often coincident but, in some, removal stops the fits Most definite in *worms*, less so, teething, adhesions of prepuce Eyes, ears nose, digestion, genitals often suggestive

5. PSYCHICAL CONDITIONS—Fright

SYPHILIS—*See* EPILEPTIFORM ATTACKS p 897

Pathology.—An epileptic attack is due to sudden discharge of nervous energy from the cerebral cortex Probably due to instability of gray matter Site of origin probably varies, illustrated by differing auræ Impulse supposed to commence in dendrites in spongy gray matter, and pass through cell to axis cylinder Subsequent path proved by cessation of spasms on one side following hæmorrhage into internal capsule in an epileptic

HISTOLOGY—No constant changes Punctate hæmorrhages in fatal status epilepticus are probably result and not cause

Symptoms.—Manifestations of attack of epilepsy are (1) Aura; (2) Loss of consciousness, (3) Convulsions, (4) Post-epileptic phenomena. Various combinations occur, loss of consciousness, partial or complete, being rarely absent.

Epilepsy—Symptoms, *continued.*

A. GRAND MAL.—

PREMONITORY SYMPTOMS.—Occasionally vague sensations for variable period, e.g., depression. Often none, and health good.

AURA.—*Is portion of the fit preceding loss of consciousness.* Often absent. Same aura usually recurs. Commonest forms are: (1) *Sensory*: Giddiness; fullness in the head; sensations commencing in the fingers, etc. (2) *Visceral*: Epigastric sensation commonest, may ascend to throat and head, when unconsciousness occurs (similar phenomenon occurs in other auræ); also cardiac and others. (3) *Special senses*: (*a*) Visual: commonest: flashes and colours. (*b*) Auditory: noises. (*c*) Smells and tastes: rare. (4) *Psychical*: Fear; Jackson's 'dreamy state' of strange surroundings. (5) *Motor*: Rare as an aura, but with onset of unconsciousness may be sudden short run or rotations.

LOSS OF CONSCIOUSNESS.—Onset sudden, often loud cry, falls without attempt at protection.

CONVULSION.—Three stages.—

1. *Tonic Stage.*—General rigidity, but severer on one side; head retracted and rotated, and usually eyes also, to one side; elbows and wrists flexed, hands clenched, or in interosseal flexion; lower extremity extended; respiratory muscles fixed, whence lividity and rapid cyanosis; pupils dilated. Duration few seconds.
2. *Clonic Stage.*—Twitching commences: progresses in severity and frequency to violent convulsion. Face, eyes, head, trunk, and limbs affected, mainly on one side. Jaw and tongue spasms cause *biting of tongue. Micturition common.* Respiration recommences noisily, and cyanosis lessens. *Frothy and often sanious saliva.* Cold sweat. Rapid pulse. Spasms diminish in frequency, often not in violence, until cessation. Duration, 1 to 2 minutes.
3. *Stage of Coma.*—Unconscious. Limbs flaccid. Congested. Deep respiration. Dilatation of pupils diminishes. Returns to consciousness gradually, or often falls asleep.

RECOVERY.—Headache, exhaustion, or slight confusion. May be vomiting, or passage of pale urine. For various sequelæ, *see below.*

REFLEXES absent during unconsciousness. On recovery, usually increased knee-jerks, ankle-clonus; plantar flexion of toes.

B. PETIT MAL.—*Transient unconsciousness, without convulsions.* Onset sudden, duration short, phenomena often slight. Expression becomes fixed, pupils dilate, slight pallor, occupation interrupted, e.g., cessation of talking or articles dropped. *Micturition common.* In some cases various automatic actions,

especially undressing. Recovery in few seconds, often unaware of occurrence.

Aura rare: occasionally previous slight faintness. Unconsciousness sometimes only partial. Sequelæ not uncommon.

Sequelæ to an Attack of Epilepsy.—

1. POST-EPILEPTIC AUTOMATISM.—More frequent in petit mal. May be no apparent loss of consciousness. Commonest form is continuation of day's work or actions without subsequent recollection of its performance. In other cases, various crimes and indecencies.
2. TRANSIENT HEMIPLEGIA, aphasia, or muscular weakness.
3. HYSTEROID CONVULSIONS.—In hysterical subjects, following petit mal.
4. STATUS EPILEPTICUS.—Recurrent convulsions without regaining consciousness. Temperature rises (103° to 105°); rapid pulse and respiration. High mortality.

Course and Occurrence of Attacks.—

RECURRENCE.—Almost an essential feature. *Frequency*: from one or two yearly to hundreds. *Remissions*: interval may be years, especially from infancy to second dentition. *Health* in intervals often good, but subjects frequently irritable or neurotic.

HOUR OF OCCURRENCE OF ATTACK.—Often constant in an individual.

NOCTURNAL EPILEPSY.—Important. Wakes with wet bed, sore tongue, slight headache and confusion. Often long unrecognized.

TYPE OF ATTACK often varies. Frequently both grand and petit mal. In others, initial petit mal develops into grand mal; or vice versa, under treatment.

Diagnosis (*see also* EPILEPTIFORM ATTACKS, p. [illegible]).—

GRAND MAL.—Characteristics: (1) Rapid unconsciousness; (2) Tonic and clonic stages; (3) Micturition; (4) Biting of tongue. Main difficulty from *hysteroid convulsions*: (1) Onset gradual; (2) Convulsions of irregular course; (3) May talk or scream; (4) Never micturates or bites tongue; (5) Long duration; (6) Rapid return to consciousness; (7) Never injured by fall.

OVER AGE OF 30 YEARS.—Investigate for an organic cause (*see* EPILEPTIFORM ATTACKS, p. 897).

PETIT MAL.—Diagnosis from: (1) Syncope. Cause often obvious; anæmia, emotion, cardiac disease, etc. (2) Auditory vertigo, Ménière's disease.

Prognosis.—Spontaneous cessation extremely rare, one attack predisposing to another.

PROGNOSIS FOR RECOVERY UNDER TREATMENT.—Unfavourable features: (1) Onset in infancy; (2) Petit mal; (3) Frequent attacks; (4) Long duration; (5) Mental weakness. Most favourable is late onset of infrequent grand mal, also nocturnal epilepsy. Pure petit mal is worse, and is often unaffected, or even aggravated, by treatment curing grand mal.

Epilepsy—Prognosis, *continued.*

No special tendency to cessation at puberty. Heredity of no influence. While any attacks continue, cessation of treatment involves aggravation.

DEATH during attack confined practically to status epilepticus or injury.

EPILEPSY AND INSANITY.—Epileptics often feeble-minded from birth. Dementia may develop, especially with: (1) Attacks frequent, over long period, and commencing in early life; (2) Particularly petit mal; insanity may commence after epileptic attacks cease ("nervous energy generated and discharge repressed"—Gowers).

Treatment.—Ascertain all details of frequency, type, and hour of occurrence of attacks. Explain necessity of prolonged treatment. Record to be kept of all attacks and treatment.

GENERAL TREATMENT.—General quiet life. Treat any ill-health, rickets, etc.

DIET.—Ordinary mixed diet. Avoid late meals. Treat gastric disturbances.

ALCOHOL.—Forbidden.

BOWELS.—Careful regulation.

EXERCISE.—Moderate. Certain forms, e.g., swimming, are obviously dangerous.

PERIPHERAL IRRITATION.—Treat carefully, e.g., adherent prepuce, worms, adenoids.

EDUCATION.—Always continue if possible, as attacks may last throughout life.

MARRIAGE.—Discourage. Great risk of epilepsy, insanity, imbecility, or neuroses in offspring. No effect on attacks if sexual intercourse moderate.

DRUG TREATMENT.—

BROMIDES are pre-eminent. Definite written instructions advisable; frequent changes to be avoided. Often control fits, sometimes cure, but when failing, other drugs rarely succeed. Administer for two years after last attack, reducing dose in second year.

DOSAGE.—For adult 30 to 60 gr. daily: maximum 90 gr. Plan large dose to precede hour of fit when known, e.g., ʒss nocte in nocturnal epilepsy; or similar dose on rising with common after-breakfast attack. Children stand bromides well.

SEQUELÆ OF BROMIDES.—(1) Acne and bromide eruption: may be obstinate. Preventive is addition to dose of liq. arsenicalis, ♏ij. (2) Mental depression. (3) Loss of appetite.

ALTERNATIVES AND ADDITIONS TO BROMIDES.—

1. Variation of bromides. Usual drug is potassium bromide: sodium, ammonium, or strontium salts may be substituted or combined.
2. Borax. Probably next best drug, gr. v to x, t.d.s.; may be added to bromides. Luminal.
3. Belladonna or digitalis may be added to bromides. Strychnine as a tonic.

PRESCRIPTIONS (dosage for adults).—

℞ Pot Bromidi gr xv Liq Arsenicalis ♏ij	Spt. Chloroformi Aq	♏x ad ℥ss
	t d s, p c.	
℞ Pot Bromidi gr xv Sod Bibornatis gr v	Tinct Digitalis Spt Chloroformi Aq	♏v ♏x ad ℥ss
	t d s, p c	

TREATMENT DURING AN ATTACK.—Place recumbent loosen clothing at neck place between teeth a tongue depressor spoon, etc Shortening of attack impossible Subsequently allow to sleep turn on side if vomiting

ARREST OF THREATENING ATTACK.—Rarely possible Occasionally by patient through effort of will Rarely with aura in a finger etc by tying tight constriction above site

STATUS EPILEPTICUS.—*Attempt to check convulsions* Chloroform inhalation most effective but fits may recur subsequently Inject chloral hydrate, gr. xxx to lx, into rectum bromides of less value Injection of hyoscine hydrobromide gr $\frac{1}{100}$ to $\frac{1}{50}$ occasionally effective (Cowen)

Tongue may fall back and need traction

JACKSONIAN EPILEPSY.

Due to irritation of motor cortex especially tumour (*see* p 854) also trauma inflammatory conditions rarely in general paralysis of the insane in uremia

Characterized by (1) Consciousness retained (or lost late) (2) Spasm commences in group of muscles (signal symptom) and spreads deliberately from limb to limb usually remains localized After many years may become generalized

EPILEPTIFORM ATTACKS.

Attacks of loss of consciousness and convulsions in adults may also occur in —

1 Chronic alcoholism
2 Syphilis General paralysis of the insane
3 Uremia and other toxemias e g lead strychnine, absinthe
4 Eclampsia of pregnancy
5 Injury to brain Post hemiplegic epilepsy
6 At onset of vascular lesions of the brain hæmorrhage, thrombosis embolism
7 Asphyxia Stokes Adams disease

VIII. INFANTILE CONVULSIONS

Convulsions resembling epilepsy, but not recurring if cause removed. Occur at age when brain still unstable If recurrent, may develop into true epilepsy

Causes.—*Debility* practically always present

(1) GASTRO INTESTINAL DISTURBANCES.

Infantile Convulsions—Causes, *continued.*

2. PERIPHERAL IRRITATION, e.g., (i) Dentition: age about 6 months. (ii) Worms, phimosis, otitis.
3. RICKETS.
4. INFECTIOUS FEVERS.—At onset: corresponding to rigors in adults.
5. ORGANIC NERVOUS DISEASES, e.g., meningitis, acute poliomyelitis.
6. EPILEPSY.

Convulsions at, or from, birth may result from injury.

Symptoms.—Tonic and mild clonic stages. Duration short: resemble mild type of an adult's grand mal. Subsequently, sleep or stuporose state. May be recurrent and fatal.

Mild 'inward convulsions', without spasm, occur in errors of feeding.

Diagnosis.—Exciting cause must be sought. Vomiting and pyrexia suggest infective or organic disease. Possibility of epilepsy increases after two years.

Prognosis.—Varies with cause and frequency of attack, and condition of patient. In severe diarrhœa, prognosis very bad. In infectious fevers, importance slight.

Treatment.—

DURING PAROXYSM.—Place in warm bath (about 96° F.): douche head with cold water if pyrexia present. If severe: (*a*) Chloroform inhalation; (*b*) Chloral hydrate, gr. iij to v in enema, preferably after rectal saline wash. If recurrent, inject morphia, gr. $\frac{1}{30}$ to $\frac{1}{25}$.

ERRORS OF FEEDING.—Wash out stomach. No emetics.

DENTITION.—Lance gums only if swollen and hot. Castor oil.

SUBSEQUENTLY.—*Treat cause.* If convulsions recurrent over a period, give bromides as in epilepsy.

IX. TETANY.

A condition characterized by symmetrical tonic spasms of the extremities, with increased irritability of the muscles and nerves to mechanical and electrical stimulation.

Pathogenesis (*see also* DISEASES OF THE PARATHYROID GLANDS).—Two factors are prominent: (1) Influence of parathyroid glands; (2) 'Toxæmia', especially of intestinal origin.

1. PARATHYROID GLANDS.—Sequels of parathyroidectomy are: (*a*) Tetany; (*b*) Increased calcium excretion. *Note*: Deficiency of calcium increases excitability of nervous system.
2. 'INTESTINAL TOXÆMIA.'—Gastro-enteritis, etc., frequently precedes tetany. As a 'working hypothesis', tetany may be ascribed to presence of a toxin, usually of intestinal origin, the removal of which needs the influence of the parathyroids; and tetany occurs when the toxin is excessive or parathyroid influence absent. Calcium in blood is diminished.

No changes present in nervous system or muscles.

Etiology.—Occurs in (a) infants, (b) adults. Both groups and all grades of severity probably of similar origin.

A. INFANTS.—Associated, almost invariably, with: (1) Rickets; (2) Gastro-intestinal disturbances, e.g., offensive stools.

Laryngismus stridulus frequently present.

Very rarely occurs in chronic constipation and helminthiasis (older children). Also in Hirschsprung's disease.

'*Carpo-pedal spasms.*'—Mild grades of tetany common: confined to hands and feet.

B. ADULTS.—

(1) DILATATION OF THE STOMACH.—Rarely in other chronic intestinal disorders and parasites. In dilated stomach, paroxysm may follow lavage, enema, or firm percussion of epigastrium.

(2) PREGNANCY, EXHAUSTING LABOUR, OR LACTATION.—Trousseau's 'nurse's contracture'. Relation to acidosis and eclampsia not yet elucidated.

(3) ACUTE SEVERE INTESTINAL INFECTIONS, especially cholera and enteric. Rarely in other specific fevers, e.g., influenza.

Removal of parathyroids in thyroidectomy a theoretical cause.

Rare instances in many affections, e.g., Graves' disease, myxœdema, syringomyelia, uræmia. Ergotism of rye is not true tetany: changes present in nervous system.

'EPIDEMIC TETANY.'—Epidemics recorded on Continent: in most instances probably secondary to other causes, e.g., diarrhœa. In Vienna outbreak tailors and shoemakers mainly affected ('shoemaker's cramp').

'FIREMAN'S' OR 'STOKER'S CRAMP'.—Intense, agonizing muscular spasms, especially of calf muscles, occur in acute diarrhœal conditions with watery stools, e.g., cholera. Frequent among stokers, probably due to draughts of cold fluid after heat of furnaces. Characteristics: (i) Numerous watery 'choleraic' stools; (ii) Intense paroxysmal muscular contractions, needing morphia injections. Prognosis good.

Symptoms.—Principal symptoms are: (1) Spasms; (2) Extremities puffy; (3) Signs dependent on increased mechanical and electrical irritability.

1. MUSCULAR SPASMS.—Are tonic, of symmetrical distribution, affect extremities mainly, and cause characteristic postures. In mild forms, confined to hands and feet.

HANDS in 'accoucheur's position': fingers flexed at metacarpophalangeal joints, extended at others: fingers pressed together and margins of hand approximated by spasm of thenar and hypothenar muscles. Thumb folded across palm, or pressed against forefinger. Wrists often flexed. *Ankles* dorsiflexed. Toes flexed.

IN SEVERE CASES.—Elbows flexed, arms crossed over chest. Knees extended. Abdomen rigid. Rarely spasm of diaphragm, with cyanosis and dyspnœa, and of pharynx. Occasionally, spasms general.

Tetany—Symptoms, *continued.*

PAROXYSMS—Onset sudden often preceded by tingling Duration few minutes to hours, or longer in adults Relaxation gradual Persist in sleep . Painful in severe cases

2 HANDS AND FEET—Swollen, tender, and hot

3 ACCESSORY SIGNS OF IRRITABILITY—Often persist after, or present without, spasm, but revealing condition of 'spasmophilia' —

a CHVOSTEK'S SIGN—Mechanical irritability of muscles and nerves A tap over trunk of facial nerve causes muscular contractions

b ERB'S SIGN—Increased irritability to electric current both faradic and galvanic, especially in ulnar nerve An anodal opening tetanus may occur

c TROUSSEAU'S SIGN—In interval between paroxysms, a spasm is induced by compression of limb or pressure on nerve trunks

Diagnosis.—Usually simple From —

TETANUS—Tetany is distinguished by (1) Onset in hands and feet (2) Posture in spasm, (3) Etiological factor

HYSTERICAL CONTRACTIONS—Generally unilateral Electrical irritability not increased

Prognosis.—Depends on associated condition

INFANTS—Serious if debilitated if diarrhœa severe or spasms widespread

ADULTS—Mortality high with dilatation of stomach

Treatment.—Indications are —

1 TREAT EXCITING CAUSE—In gastric dilatation lavage if spasms severe gastrojejunostomy is recommended but mortality high In pregnancy etc parturition or weaning stops spasms Parathyroid extract at present valueless

CALCIUM SALTS—Give calcium lactate, gr xv four hourly, and food rich in calcium, viz, eggs and milk (Effects unproved)

2 RELIEVE SPASMS—Warm baths In children chloral gr. iij to v. by rectum For spasm of the larynx chloroform anæsthesia Light diet Fresh air In adults, chloral and other sedatives, e g —

℞ Sodii Bromidi gr xx | Aq Chloroformi ad ℥j
Tinct Valer Ammon ♏xxx to xl |
Every four hours while spasms last

CONVALESCENCE—Treat rickets, digestion, etc Tonics.

X. PERIODIC PARALYSIS.

A rare familial and hereditary disease characterized by recurrent attacks of flaccid paralysis

Etiology.—Familial and hereditary Both sexes affected. Attacks commence about puberty. *Exciting causes* doubtful: may be

over-exertion, emotion, gastric disturbances, or toxæmia (certain articles of food). No pathological changes. *Pathogenesis* is unknown: origin probably in muscles. Migraine may occur in family.

General Description.—Recurrent attacks of transient flaccid paralysis. Prodromata absent, or slight aching. Paralysis commences in legs, often at night: spreads to arms and trunk: usually complete in twenty-four hours. *Recovery* begins within twenty-four to forty-eight hours: reverse order to onset. Cranial nerves rarely, and diaphragm very rarely affected. No mental, sensory, or sphincter changes. Temperature normal. Reflexes absent. Heart may be dilated. *Recurrences*, usually one to two weeks' interval: may cease in later life. Death in attack, rare. Creatinine excretion diminished during attack.

Treatment.—Potassium citrate recommended. During attack, treat failure of respiration or heart.

A recurrent oculomotor and a recurrent facial paralysis also exist.

CHAPTER CXXXV.

PSYCHONEUROSES.

I. HYSTERIA.

A condition in which ideas control the body, and produce morbid alterations in its functions.

Etiology.—

AGE.—Commonest between 15 and 30 years. Rarely from 8 to 15 years. When established, may persist throughout life, mainly in women.

SEX.—Females predominate. Very rare in adult males.

HEREDITY.—Of greatest importance, a neuropathic family history. Additional factor is deficient training and control in childhood; often further influenced by neuroses in the mother.

RACE.—Latin, Jewish, and Slav races are specially susceptible. Major convulsions very rare in other races.

EXCITING CAUSES.—Shocks, especially psychical, e.g., love affairs, fright. The necessary degree of shock varies with the instability of the subject.

Theories of Hysteria.—No organic disease of the nervous system present. Theories are numerous, mostly of great complexity, and need reference to special works.

1. PSYCHOSIS (Charcot).—The body is controlled by ideas.
2. BABINSKI.—Manifestations are due to auto-suggestion. Various impressions are excluded from the patient's consciousness, e.g., from a certain limb; in extreme forms even dual personality, each identity being unaware of the other. Thus differing from dual personality in psychasthenia which is recognized by the subject.

Theories of Hysteria, *continued.*

3 FREUD —Sexual activities, often perverse, occur mentally in the period before puberty, constituting mental traumata These may be repressed to the subconscious mind, where they remain unneutralized, though forgotten by the conscious mind Resuming activity later, in certain circumstances, these sexual mental traumata, though they may still remain subconscious, influence the conscious mind, and produce hysterical manifestations

Freud thus refers all hysteria to a sexual origin as the Greeks of old By 'psycho analysis' and study of dreams, the physician patiently drags out the original skeleton, exhibits it, and lays the ghost

Symptoms.—Of every degree and variety May simulate practically every organic disease of the nervous system and many of other systems A summary is given first, and then certain additional details Often many varieties co exist, or follow in same individual

SUMMARY.—

A CONVULSIVE FORMS —(1) Minor hysteria (2) Major hysteria (hysteroepilepsy)

B NON CONVULSIVE FORMS —

1 *Psychical Forms*

2 *Motor System* —(*a*) Contractures and spasms, (*b*) Paralyses, (*c*) Tremors (*d*) Spasmodic movements

3 *Sensory System* —(*a*) Pain of every variety, (*b*) Anæsthesia, (*c*) Hyperæsthesia, (*d*) 'Hysterogenic spots'

4 *Special Senses* (*a*) Restriction of field of vision (*b*) Deafness, or extreme sensitiveness of hearing, (*c*) Absence of smell or taste, or extreme sensitiveness

5 *Alimentary System.*—(*a*) Dyspepsia of every variety (*b*) Diarrhœa, constipation, vomiting (*c*) 'Phantom tumour' (and pseudo cyesis) Rarely (*d*) Anorexia nervosa, (*e*) Ileus, irritable rectum, anospasm

6 *Respiratory System* —(*a*) Rapid respiration, (*b*) Cough, (*c*) Hiccough, yawning, (*d*) Hæmoptysis

7 *Cardiovascular System* —(*a*) Tachycardia, (*b*) Pseudo angina, (*c*) Flushing, sweating

8 *Joint Affections*

9 *Sphincter Affections*

10 *Pyrexia*

MOST CONSTANT SYMPTOMS FOR GENERAL DIAGNOSIS OF HYSTERIA —

1. *Psychical symptoms* —Rarely absent Especially, laughing and crying attacks, fainting, emotions, 'globus hystericus'

2. *Anæsthesia.*—(i) 'Glove' or 'stocking' type, (ii) Hemianæsthesia

3 *Fields of vision constricted* with progressive spiral diminution.

4. Exaggerated symptoms with negative physical signs, temperature normal, plantar reflex flexor.
5. 'Clavus hystericus.' Pains in back. Heart complaints. Retention of urine.

A. CONVULSIVE ATTACKS.—

1. MINOR FORMS.—Preceded by emotional disturbance (*see* PSYCHICAL FORMS). Then irregular clonic movements. Falls without injury. Does not bite tongue, or pass water. Becomes 'unconscious'. Gradual recovery with much emotional display: often passes flatus or much pale urine. Subsequently, hazy recollection of occurrence. Torpor or catalepsy may follow.
2. MAJOR FORMS (*Hystero-epilepsy*).—Mainly in Latin races. Very rare in British Isles and America. Four stages described—preceded by emotional disturbances:—
   1st stage.—Epileptoid convulsions, clonic and tonic.
   2nd stage.—'Phase de grandes movements.' Various contortions, screaming, etc.
   3rd stage.—'Phase des attitudes passionelles.' Attitudes expressing emotions, beatitudes, erotic, etc.
   4th stage.—Return to consciousness. Hallucinations, visions and conversations described and may subsequently be believed.
   Duration.—Fifteen to thirty minutes.

B. NON-CONVULSIVE ATTACKS.—

1. PSYCHICAL FORMS.
   i. *Acute Mild Forms.*—Alternate laughing and crying. 'Globus hystericus' and constriction in throat. Fainting, excitement, and emotions (rarely, may pass into mania).
   ii. *Severe Forms.*—(a) Trance. (b) Catalepsy: limbs flaccid but remain in any position in which placed: trance co-exists. (c) Status hysterious: in bed for months oblivious to all, breath foul, delirium: may be suicidal.
   iii. *Chronic Forms.*—Desire for sympathy leads to exaggerated symptoms, to self-inflicted wounds, to long-continued deceptions bordering on malingering.
2. MOTOR SYSTEM.—
   Spasmodic contractures.—Onset spontaneous, or following emotion, pain, fit, or injury. Spasm powerful, increased by efforts to relax, often persists in sleep, relaxed under anæsthetics; often disappears suddenly even after long duration; may recur.
   Distribution: Monoplegia (hysteria being commonest cause of such), arm or leg, latter simulates lateral sclerosis. Also hemiplegia, paraplegia, ptosis, trismus, and 'phantom tumours'.
   Paralyses.—May simulate any organic disease. Characters: (i) Paralysis rarely absolute, e.g., unable to stand or walk, but free leg movements in bed ('astasia abasia'). (ii) Movement opposed by contraction of antagonistic

Hysteria—Symptoms, *continued.*

muscles (occurs only in hysteria). (iii) No wasting of muscles. (iv) Anæsthesia common. (v) Reflexes increased; plantar reflex flexor; pseudo ankle-clonus common. (vi) Electrical reactions normal. Duration: transient or for years. Later: atrophy from disuse, tendon contractions, and joint changes.

Distribution: (*a*) Paraplegia, commonest. (*b*) Hemiplegia: tongue may deviate towards affected side: hemianæsthesia usual. (*c*) Monoplegia: usually with 'glove' or 'stocking' anæsthesia. (*d*) Larynx: adductors of vocal cord, very common, aphonia or whispering; often cured by examining larynx, or by electric current. Other cranial nerve distributions rare.

Tremor.—Common. Type varies: usually fine tremor of hand. Increased by voluntary movements. May be 'intention tremor', whence early disseminated sclerosis often diagnosed as hysteria.

Spasmodic movements.—May be: (*a*) Irregular, as in chorea. (*b*) Repeated, as in 'habit spasms', or rhythmical: sometimes a single muscle, e.g., psoas. (*c*) Rarely, complex and purposive, e.g., salaaming.

3. SENSORY SYSTEM.—

Pain of every variety. Commonest: 'clavus hystericus' (nail driven into head), and in back. Simulates many diseases, e.g., caries of spine, appendicitis, gastric ulcer: pseudo-physical signs increase difficulty.

Anæsthesia—Very common. Must be looked for: patient often neither complains nor knows. Usually complete to all sensations, but occasionally 'dissociation'. Includes deep structures and mucous membranes within area. Bleeding slight on pricking. Muscular sense often preserved, e.g., sewing. 'Allochiria' occasionally: touch, etc., referred to other sites. Duration: may be years, yet disappear suddenly. On treatment, may change sides and then revert.

Distribution: (i) Hemianæsthesia: sharply limited at mid-line, includes palate; conjunctiva escapes usually. (ii) 'Glove' or 'stocking' anæsthesia of limbs. Sharp circular line of demarcation, i.e., corresponds to no nerve or root distribution.

Hyperæsthesia.—In various areas. May be to light touch or deep pressure.

'Hysterogenic Spots.'—Common sites of symmetrical hyperæsthetic spots are ovarian, inframammary, and over dorsal spines. Extreme tenderness. Pressure often induces other hysterical symptoms.

4. SPECIAL SENSES.—

Sight.—Fields of vision frequently affected. Characters: (i) Field constricted. (ii) As perimeter observations are

continued, *field diminishes in a spiral* (pathognomonic). (iii) Reduction greatest for blue and least for red (contrary to organic disease). (iv) With hemiplegia, constriction of field is often on the same side, crossed amblyopia. Blindness rare. Excessive sensitiveness to light.

Hearing.—Deafness, or excessive sensitiveness to sound (hyperacusis).

Absence of taste and smell: very common.

5. ALIMENTARY SYSTEM.—

Dyspepsia of various types. Appetite failing; hyperchlorhydria; difficulty in swallowing and regurgitation from spasm of œsophagus. Fasting is often fraudulent. Flatus and borborygmi common ('peristaltic unrest').

Diarrhœa, often very resistant, of lienteric type. Constipation common. Vomiting common; rarely fæcal.

'Phantom tumours' in abdomen. Result from spasm of diaphragm with relaxation of abdominal muscles, intestinal distention with gas, and arching of vertebræ. Simulate tumours or pregnancy, especially at menopause ('pseudo-cyesis'). Relax under anæsthetics.

Anorexia Nervosa.—Rare. Characterized by: neuropathic history, great antipathy to food, most extreme emaciation. Often fatal, but recoveries occur at any stage. Other hysterical symptoms often absent.

Ileus. Irritable anus. Anospasm.

6. RESPIRATORY SYSTEM.—

Rapid respiration. Deep breaths.

Cough. Especially 'barking cough of puberty'.

Hiccough; yawning.

Hæmoptysis: usually from pharynx. Simulates phthisis.

7. CARDIOVASCULAR SYSTEM.—

Tachycardia, common.

Complaints of præcordial pain. Pseudo-angina (*see* p. 708).

Flushing, sweating.

'Stigmata' or hæmorrhages into the skin are mainly if not invariably fraudulent.

8. JOINTS.—Usually single large joint affected, hip or knee. Painful, with wide superficial and deep tenderness, muscles contracted: may be trophic changes, some œdema and warmth. No real shortening; no changes in radiogram; normal under anæsthetic. Often cured by 'quack' methods.

9. SPHINCTERS.—Retention common. *Never incontinence*, except by overflow. Passage of much pale urine common.

10. FEVER.—Temperature practically always normal. Pyrexia in rare instances. Hyperpyrexia repeatedly proved fraudulent.

Prognosis.—Liability to hysterical manifestations persists for many years, usually diminishing after age of 30 to 35 years.

In a given symptom, e.g., paralysis, duration cannot be foretold:

Hysteria—Prognosis, *continued*

after existing many years may disappear suddenly, often following a shock. No symptom is necessarily permanent.

Anorexia nervosa and, very rarely, persistent vomiting are the only hysterical manifestations with definite mortality.

Diagnosis.—Inquire into previous hysterical symptoms, family history, and make *complete physical examination.* Of the most constant symptoms (*see* SUMMARY *above*), several are almost invariably present, and make diagnosis simple.

DIFFICULTIES.—

1. EXCLUSION OF ORGANIC DISEASE OF NERVOUS SYSTEM.—Diagnosed as hysteria are: (i) *Early disseminated sclerosis,* frequently; (ii) Intracranial tumour, occasionally.
2. PRESENCE OF BOTH HYSTERIA AND ORGANIC DISEASE.—Possibility always to be considered.
3. DISTINCTION OF HYSTERIA FROM PURE MALINGERING.

Treatment.—The physician's responsibilities include: (1) Treatment of the patient. (2) Choice of a nurse. (3) Treatment of the patient's relatives. Relatives frequently have also hysterical taint; are over-sympathetic, or, per contra, bully the patient; are partly responsible for condition; and difficult to deal with: hence advisable, and often essential, to remove patient from home. To the patient physician must never give impression that he considers that she is malingering, and that "it is her own fault"; he should give an explanation of condition and probability of recovery; and must gain her confidence. Treatment necessarily varies with each symptom and patient.

MILD FORMS.—Change of scene. General health attended to. *Aperients.* Later: general tonics and *suitable occupation.*

SEVERER FORMS.—Isolation in bed usually necessary for varying periods. Forms of treatment include:—

HYDROTHERAPY.—In minor psychical forms, in fits and spasm, cold water applied with apparent disregard for clothing frequently effective, or a cold bath. In more chronic conditions, cold spinal douche valuable.

ELECTRICITY.—Strong faradic current (harmless pain) frequently arrests convulsions.

MASSAGE.—Of value for general nutrition.

COUNTER-IRRITATION, BLISTERS.—Often effective by suggestion.

DRUGS.—*Morphia always to be avoided.* Hypodermic injections of water often equally effective for sleeplessness.

Narcotics.—Avoid if possible. Cachets of sugar often effective.

Valerian and *Asafetida.*—Valuable, especially in chronic minor forms, e.g., dyspepsia.

℞ Tinct. Valer. Ammon. ♏xxv | Aq. Camph. ad ℥ss
Tinct. Asafetidæ ♏xxv

Bromides.—Of great value. May be combined with last.

SPECIAL METHODS OF TREATMENT.—

(1) WEIR-MITCHELL.—Isolation: massage: large quantities of

milk. (*See* NEURASTHENIA.) The relaxation of each restriction should depend on improvement, and be re-imposed on relapse; the condition being explained to patient.

2. HYPNOTISM.—Inadvisable.
3. SUGGESTION.—Good results with selected cases in reliable hands.
4. FREUD'S METHOD.—'Psycho-analysis.' Aim is to elucidate an original cause for hysterical manifestations, these being, according to Freud, of sexual origin. Method is complex, and theory and result still under trial.

NEURASTHENIA.

A functional condition in which exhaustion of the vitality of the nervous system causes inefficiency of the mind and body. The 'vital force' of the nervous system is under-engined for the normal stress of life, either hereditarily, or from some exceptional strain to which the individual has been subjected.

Etiology.—

AGE.—Usually 25 to 50 years.
SEX.—Commoner in males.
HEREDITY.—Born neurasthenics are common.
PROLONGED MENTAL WORRY.
SPECIFIC DISEASES.—Especially influenza (even mild) and severe enteric.
DRUGS.—Cocaine, morphia, alcohol; but drugging often results from neurasthenia.
TRAUMA.—Special type (*see* TRAUMATIC NEUROSES, p. 910).
SEXUAL FACTORS.—Influence and mode undecided.

Symptoms.—Very varied. Certain common basic symptoms, usually with accentuated disturbance in various systems, constituting different types, viz., psychical (or cerebral), motor (or spinal), gastric, sexual, cardiac and other visceral forms. Distinction of types often over-exaggerated.

GENERAL SYMPTOMS AND CONDITION.—Common phenomena:—

APPEARANCE.—Often characteristic of depressed bodily and mental vigour, of tiredness and despondency, with pinched facies of vasomotor disturbance.
LOSS OF WEIGHT.
PALLOR and some anæmia usual.
SUBJECTIVE SYMPTOMS MARKED, with slight objective signs. Described by subject in over-full detail.
RESTLESSNESS.—*Worried by trifles.* Irritable, despondent, and egotistical.
HEADACHE.—Often vertical oppression. Vague sensations common, e.g., 'brain feels too big for the head'.
PAINS IN BACK.
INSOMNIA or unrefreshing sleep.

Neurasthenia—Symptoms, *continued.*

HYPERÆSTHESIA.—From tinglings, formication, etc., to pains in various sites.

PSYCHICAL OR CEREBRAL FORM.—'Anxiety neurosis'. Loss of power of concentration and mental work. '*Phobias*' very common. Frequent fear of death, insanity, poverty, etc. Various severe forms, e.g., (1) Agoraphobia, fear of open spaces; (2) Claustrophobia, fear of closed rooms. Other symptoms in this group: Restlessness, bodily and mental. Involuntary mental activity: thoughts run rapidly through the head.

MOTOR OR SPINAL FORM.—Muscular weakness, may be extreme. Pains in back and limbs. Tender spots on spine not uncommon. Hyperæsthesias and visceral neuralgias common. Muscles flabby; often fine tremor of hands; may be some inco-ordination.

SPECIAL SENSES.—Often disturbed, especially vision. Eyes tire rapidly (errors of refraction common). Hyperacusis.

CIRCULATORY SYSTEM.—Important and common changes. *Vasomotor Disturbances.*—May be: (1) Peripheral vessels contracted: extremities blue, pinched facies, desire for warmth. (2) Peripheral vessels relaxed: (i) Arterial pulsation marked, especially abdominal aorta. (ii) Capillary pulsation; (iii) *Pulse* almost water-hammer. Other signs are: (3) Flushing or blushing frequent; profuse sweating (may be nocturnal).

GASTRIC AND GASTRO-INTESTINAL FORM (*see* GASTRIC NEUROSES AND MUCOMEMBRANOUS COLITIS).—Constipation, poor appetite, and flatulence common in all forms.

SEXUAL FORM.—Some complaint of sexual functions almost invariable. Spermatorrhœa common: frequent nocturnal emissions, or sometimes after defæcation. Other complaints are fear of impotence, presence of nervous impotence, 'irritable testis', aching in pelvis or genitals, and in women, tender ovary or dysmenorrhœa.

CARDIAC FORM.—Palpitations, præcordial sensation, rapid heart-beat, often dizziness. Characterized by abnormal increase of heart-rate on slight exertion. Vasomotor disturbances, as above, common. Occasionally pseudo-angina. Other signs of neurasthenia may be slight.

SENSORY SYSTEM.—Tingling, formication, hyperæsthesia, etc.: pain in various sites.

URINE.—Often scanty, with increased urates, oxalates, or sometimes phosphates. Micturition may be frequent. *Never incontinence.*

ON EXAMINATION.—No signs of definite organic disease. *Reflexes* increased or normal. Knee-jerks increased; plantar reflex flexor; no ankle-clonus. No definite paralysis. No Romberg sign, but swaying often exaggerated. *Pupils* dilated, rarely unequal, reactions normal. Errors of refraction common. No alteration of the field of vision.

Diagnosis.—From two groups of conditions: (a) Organic diseases, especially tabes, dementia paralytica, and the rare myasthenia

gravis; (b) A chain of psychoses and neuroses, from hysteria to the borderland of insanity. Examination should always be complete; Wassermann reaction advisable. Serious organic disease of any kind, e.g., cancer, may suggest neurasthenia.

TABES.—Resembles spinal form of neurasthenia. Differs in reflexes and pupil changes.

DEMENTIA PARALYTICA.—May commence like neurasthenia. Note: impaired memory, defects in articulation, pupil changes, cerebrospinal fluid.

HYPERTHYROIDISM AND EXOPHTHALMIC GOITRE may resemble cardiac form.

PSYCHASTHENIA.—A group including many cases akin to, and formerly classified as, psychic or cerebral neurasthenia. Onset in youth; hereditary factor; persists through life with remissions. Main features (Janet) are: (1) Certain *stigmata* of indecision: (a) Inability to concentrate attention, doubts, hesitation, even feeling of dual personality (*see* HYSTERIA, p 901); (b) Physical: clumsy movements, tics. (2) *Obsessions* of all kinds, from minor grades to kleptomania, crime, and sexual acts. (3) *Imperative ideas or acts*: tics, phobias. General neurasthenic symptoms may be present Reaches borderland of insanity; but no delusions, hallucinations, or impairment of memory.

HYSTERIA.—Diagnosis by stigmata: anæsthesia, restriction of visual fields, contractures, convulsions. Often difficult

HYPOCHONDRIASIS.—Conviction that sensations are due to organic disease. Actual delusions occur.

Prognosis.—*Recovery* never rapid, recurrences common; but cures may be effected and great improvement can be promised with proper treatment. Favourable factors in prognosis: (1) Patient's circumstances permitting treatment (2) Removable cause; (3) Short duration; (4) Previous health good and no hereditary neurosis

Treatment.—Make certain of absence of organic disease, and reassure patient.

Indications are: (1) Remove the cause, (2) Rest and restore the nervous system. Plans must be adapted to the patient, his story carefully heard, and his confidence gained.

REST.—In cases of worry and overwork, a prolonged rest and absence—at least six months. In severer forms, a nurse and a daily routine. In most severe cases, 'Weir-Mitchell treatment' or 'rest cure': principles being: (1) Prolonged rest in bed away from home and friends, at least six weeks; (2) Abundant simple diet beginning with milk; (3) Massage. Results often excellent, sleep returning, weight increasing, and nervous system calming.

BOWELS.—Regulate motions.

PERIPHERAL IRRITATION, local disease, and septic foci must be searched for and treated, e.g., errors of refraction, anæmia, gastric or intestinal disturbances, movable kidney, genital and pelvic diseases, oro-nasal infections.

Neurasthenia—Treatment, *continued.*

DRUGS.—Of subsidiary value except for special symptoms.

GENERAL TONICS.—Arsenic, iron, strychnine, glycerophosphates.

SEDATIVES when pains severe: bromides, phenacetin, aspirin. Withdraw when possible. Avoid alcohol, morphia, and chloral hydrate.

HYDROTHERAPY.—Often of great value. Wet packs, douches, or elaborate methods of spas. *Electricity* may well be combined.

PSYCHOTHERAPY.—Suggestion and its various developments, and, in some cases, Freud's psycho-analysis (*see* HYSTERIA), have effected many cures, with selected cases, in proper hands.

INSOMNIA.—Avoid drugs if possible. Hot drink or a little food; wet packs; Weir-Mitchell treatment. Trional and sulphonal if necessary.

PROPHYLAXIS.—Neurotic children need careful watching; protection from educational strain, and special attention at puberty. When they become adults, should have regular holidays. Exercise and fresh air of great value, but strength not to be overtaxed.

III. TRAUMATIC NEUROSES.

(*Traumatic Neurasthenia. Railway Spine.*)

A group of conditions following shock, bodily, mental, or both, with symptoms of neurasthenia, hysteria, and various psychoses.

Etiology.—May follow: (1) Mental shock; (2) Concussion or accidents involving bodily injury; (3) Concussion or accidents without bodily injury. Mental shock is included in the latter groups.

Symptoms.—Several groups. Normal excitement, of few days' duration, immediately following above events, is not included.

1. *Traumatic neurasthenia.*—Interval of days or weeks usual between cause and onset of symptoms. Cause generally includes concussion (groups (2) and (3) of ETIOLOGY). Symptoms of ordinary neurasthenia, often of spinal form ('railway spine').
2. *Groups with symptoms of hysterical or mental nature, or psychoses.*—Condition immediately follows cause, which involves marked mental shock. Symptoms various: headache, apathy, loss of memory, emotional states, etc., in various combinations. Hysterical anæsthesia not common; restriction of visual fields more frequent. (Includes 'shell-shock'.)

Prognosis.—(1) In simple traumatic neurasthenia the prognosis is good. In claims for compensation, recovery is unusual before conclusion of litigation; if interval has been lengthy, recovery not invariable even if action successful. (2) The second group is frequently very resistant to and needs prolonged treatment; various psychoses, melancholia, delusions, and dementia may develop.

Diagnosis.—From: (1) *Malingering*, especially in neurasthenic group. May need considerable observation. (2) *Definite injury and organic lesions of nervous system.* Examine for signs of cord and brain injury and bladder troubles. X rays.

IV. TICS: HABIT SPASMS.

A tic ('twitch' or 'jerk') is a co-ordinated purposive act often performed originally for a reasonable cause, but the repetition and persistence of which is due to a psychical disorder. Thus, a head tic may arise from irritation of a frayed collar; is reasonable while the cause is present; but its persistence only occurs with, and is due to, a neuropathic state: and further, it may arise without any known stimulus.

RELATION TO 'SPASMS'.—A 'spasm' is a motor reaction resulting from irritation at some point in a reflex spinal arc or bulbo-spinal arc, is independent of consciousness or the will, and has no psychical factor.

Certain conditions are as yet undetermined as 'spasms' or 'tics': e.g., facial spasm, spasmodic torticollis.

Nomenclature is confused: thus 'habit spasm' is a tic; movements in 'tic douloureux' are spasms; 'chorea major' is hysteria.

RELATION TO HYSTERIA.—Tics merge into hysteria, especially severer tics, viz., saltatory spasms, chorea major.

RELATION TO INSANITY.—Psychical tics and obsessions merge into monomania.

RELATION TO SYDENHAM'S CHOREA.—None. No relation to acute rheumatism or endocarditis.

Etiology.—

AGE.—After early childhood at any age, especially puberty. Not under 4 years.

SEXES.—Equal.

HEREDITY.—Subjects often clever, but neuropathic taint.

PREDISPOSING CAUSES.—Debility or mental strain. Peripheral irritation, e.g., blepharospasm from conjunctivitis. Mimicry occasionally. Rarely follows Sydenham's chorea.

Morbid Anatomy.—No changes.

Groups of Tics.—(1) Simple tics or 'habit spasms': common. (2) Co-ordinate tics: rare. (3) Convulsive tics: very rare. (4) 'Psychical tics': not common. (5) Various conditions allied to, and sometimes described as, tics: spasmodic torticollis, facial spasms, saltatory spasms ('jumpers'), chorea major, latah.

1. **Simple Tics or 'Habit Spasms'.**—

MOVEMENTS.—(1) Limited usually to small group of muscles. (2) Under control of will to some extent: attempt to restrain is severe mental effort, often followed by specially severe tic and depression at failure. (3) Later become habitual and unconscious. (4) Cease in sleep; increased by excitement. (5) Same movement

Tics, *continued.*

is repeated; intermissions complete (6) Often of *extreme rapidity*; less commonly, slow and deliberate (usually larger tics). (7) Always co-ordinated; purposive in character, but causeless and resultless.

VARIETIES OF TIC.—Innumerable: especially of face and head. Frequent are: twitchings of mouth or eyebrows; blinking, often with jerks of head; shrugging of shoulders; sniffing (respiratory tic). Lower limbs less common.

PROGNOSIS DEPENDS UPON:—

AGE.—With onset in childhood, often cease; onset in adults, often permanent.

DURATION.—The longer it has lasted, the more difficult it is to cure.

MENTAL CONDITION AND NEUROPATHIC FAMILY HISTORY.

CAUSE.—Arising from definite stimulus (peripheral irritation, ill health) better than causeless onset. Shock occasionally arrests tic, permanently or temporarily.

No effect on duration of life.

DIAGNOSIS.—By characteristics of: (1) Repetition; (2) Complete intermission; (3) Purposive; (4) Co-ordinate; (5) Extreme rapidity (usually).

CHOREA.—Purposeless; not repeated; cure comparatively rapid; relation to rheumatism.

HYSTERIA.—Movements may be identical, but other stigmata present, e.g., globus, anæsthesia, contraction of visual fields.

REFLEX SPASMS.—Difficult. Confined to some definite nerve distribution.

TREATMENT.—

GENERAL.—Remove any irritation, e.g., adenoids, ocular defects, prepuce. Avoid overstrain mentally, and ensure mental rest. Suggestion, in severe forms.

MOVEMENTS.—Subject stands motionless before mirror, at first for few seconds, then longer: persist for weeks after cessation of tic.

EXERCISES to use affected and antagonistic muscles rationally.

DRUGS.—Arsenic (as in chorea). Tonics. Sedatives: Liq. ext. of conium ♏v, t.d.s. (increasing ♏j alternate days), with pot. brom. gr. v, t.d.s.

2. **Co-ordinate Tics.**—Applied to complex tics involving complex movements, otherwise no distinction from simple tics.

Note.—All tics are co-ordinated movements.

3. **Convulsive Tics** (*Gilles de la Tourette's Disease*).—

AGE.—Usually in children, rare after puberty. Neuropathic family history generally marked. Condition borders on insanity. *Four characteristics*: some or all may be present together:—

1. MUSCULAR CONTRACTIONS.—Movements as in simple tics, but greatly exaggerated; occur in attacks; repeated irregularly.

ii. EXPLOSIVE UTTERANCES.—Irrelevant words; oaths ('coprolalia'). Occur with or before movements.

iii. IMPULSES OF MIMICRY.—Echolalia or echokinesis (mimicry of actions).

iv. MENTAL 'OBSESSIONS': 'PSYCHICAL TICS'.—Repetition of a certain word, action, or number before performing any action.

TREATMENT.—Rest of body and brain. Suggestion. Massage. Baths. Electrical treatment.

4. **'Psychical Tics.'**—Innumerable varieties of obsessions: e.g., adult avoids stepping on line between flagstones. No movements occur. Allied to hysteria and monomania, but regardable as 'tics of the brain'. Famous instances occur amongst the world's greatest men.

TREATMENT.—Often incurable: many are harmless: may drift into insanity. General treatment and suggestion.

5. **Various Allied Conditions sometimes described as Tics.**—

SPASMODIC TORTICOLLIS.—*See* p. 915.

FACIAL SPASM.—A spasm and not a tic. (*See below.*)

SALTATORY SPASMS ('Jumpers').—Described as 'contraction of muscles of lower limbs occurring when soles are placed on the ground', viz., 'jumpers'. In men and women with neuropathic taint: may be epidemics. Usually transitory, sometimes lasts for years.

CHOREA MAJOR.—True hysteria. Various dancings and movements occurring as epidemics in religious excitement of middle ages.

LATAH.—A special psychosis of Java and Borneo. The subject is compelled to perform any action dictated by any person. Usually persists through life.

V. FACIAL SPASM.

Spasms confined to muscles supplied by the 7th nerve fall into two groups:—

1. ORGANIC DISEASE OF NERVOUS SYSTEM.—Irritation of cerebral cortex; or compression of nerve trunk by tumour, etc., at base of brain.
2. IDIOPATHIC FACIAL SPASM.—No organic disease.

Spasm of facial muscles also occurs in many conditions not to be considered as 'facial spasm', e.g., chorea, epilepsy, hysteria, habit spasms and tics, tetanus, tetany, athetosis; also in muscles paralyzed in previous Bell's palsy.

Idiopathic Facial Spasm.—

ETIOLOGY.—Age 45 to 60 years. Females commoner than males. No heredity. Often no exciting cause. Sometimes peripheral irritation, carious teeth, etc.; in others emotion or shock.

When established, paroxysms often excited by cold, draughts, emotion, voluntary movements.

Idiopathic Facial Spasm, *continued.*

SYMPTOMS.—Spasms usually *unilateral, clonic, occurring in paroxysms, and without paresis.* Occur in all degrees of severity and range. At onset often slight and occasional, later becoming severer.

IN TYPICAL FORMS OF SEVERER CHARACTER.—Paroxysm commences with slow contractions of limited range, becoming faster and faster and more diffuse until a tonic contraction occurs: passes off with diminishing contractions but usually of wider range.

DISTRIBUTION OF SPASM.—Orbicularis palpebrarum and zygomatic muscles most commonly affected. Severe attack usually commences there. All muscles may be affected, including platysma and stapedius. Severe attack may spread to opposite side.

SENSATION AND ELECTRICAL REACTIONS.—Unchanged.

PARTIAL FACIAL SPASMS.—Extremely common, especially blepharospasm (eyelids). Blepharospasm may be: (1) Clonic, rapid winking; also in tics and hysteria. (2) Tonic, usually a reflex with photophobia; eyelids closed for several minutes. Spasm may be very limited, fibrillary twitching of muscles ('live blood in the eye').

COURSE AND PROGNOSIS.—Severer forms often intractable, and when ceasing, relapses common. No effect except mental depression.

DIAGNOSIS.—By characteristics of (1) persistence, (2) paroxysms, (3) absence of paralysis. Diagnosis from (*a*) organic disease, (*b*) other conditions of spasm.

ORGANIC DISEASE.—Some paralysis or paresis present

HYSTERIA.—Spasm tonic. Other stigmata.

TICS.—Some voluntary control. Not limited to 7th nerve distribution.

REFLEX FROM PERIPHERAL IRRITATION.—Often tenderness of 5th nerve trunks on pressure.

TREATMENT.—*Indications*: (1) Ascertain cause; (2) Remove any peripheral irritation.

GENERAL HYGIENE.—Maintain general health; avoid draughts, cold, and stimuli.

LOCAL TREATMENT.—Counter-irritants: hot fomentations or blisters to face, back or neck, or behind the ear. Massage of affected muscles. Electric treatment of little value.

DRUGS.—Little value. Tinct. gelsemii ♏ xv, t.d.s., or ext. conii liq. ♏ v, t.d.s. Avoid morphia.

MILD TYPES (e.g., 'live blood in eye').—Usually yield to general treatment, local light massage, bathing, and gentle pressure at supra- or infra-orbital foramina.

SEVERE TYPES.—Frequently intractable. Special measures: (1) Schlösser's treatment: injection of alcohol into nerve at stylomastoid foramen, producing facial paralysis. Spasm returns as paralysis passes, but usually several months' relief, and can be repeated. (2) Operation: division of nerve and anastomosis with spinal accessory.

VI. SPASMS OF THE MUSCLES OF MASTICATION.

Spasm may be : (1) Tonic ('trismus') ; or (2) Clonic. Usually part of a general condition : less often of local origin.

1. **Tonic Spasm** ('*Trismus*' or '*Lock jaw*').—Inability to separate teeth. Occurrence :—

GENERAL CONDITIONS.—Tetanus. Epileptic fit, tonic stage. Rarely in hysteria and tetany.

LOCAL CONDITIONS.—Protective spasm or inflammation of muscles from carious teeth, gingivitis, mumps, or from cold.

Very rarely, in lesions of nucleus in pons, or irritation of nerve in basal meningitis.

Distinguish from osteo-arthritis and disease of jaw-joint.

2. **Clonic Spasm** ('*Chattering teeth*').—Rigors. General convulsions. Cold.

VII. SPASMODIC TORTICOLLIS.

Spasm of the muscles of the neck, affecting position of head. No organic changes in the nervous system. The clonic type is a true tic.

Etiology –

AGE.—Adults.

SEXES.—Equal: apparent excess in females is due to hysterical spasms.

PREDISPOSING CAUSES.—Neuropathic taint.

EXCITING CAUSES.—Debility ; cold ; disorders of vision ; local injury. Often none.

Symptoms.—

ONSET.—Gradual. Increases in frequency and extent. Rarely sudden.

CHARACTER OF MOVEMENTS.—Two types. (1) Clonic : 'jerks'. At onset, occurs at long intervals : finally may be 20 to 30 per minute. Very distressing. (2) Tonic position of head long maintained. Both types may occur in same patient.

As in other 'tics', initially under control of will, but effort exhausting. Tic is preceded by feeling impelling movement. Ceases in sleep. Increased by emotion. Sometimes controlled by antagonistic movement, e.g., finger pressed under chin. Discomfort considerable, rarely great pain.

MUSCLES.—Never waste ; may hypertrophy. Electrical reaction normal.

TYPE OF MOVEMENTS.—

1. STERNOMASTOID CONTRACTIONS.—Commonest form : generally on right side. Draws mastoid towards shoulder, turning head to opposite side and raising chin. Usually associated, as disease progresses, with other muscles, e.g. : (*a*) Trapezius, upper part, movement similar ; (*b*) Splenius of opposite side, tilts head backward. Arm (opposite side) or face occasionally affected.

Spasmodic Torticollis—Symptoms, *continued*

2 'RETRO-COLLIC SPASM'—Deep posterior neck muscles Head drawn back. forehead wrinkled and eyebrows raised, from occipito frontalis contraction

Rarely: Anterior neck muscles Chin on chest

Occasionally other muscles complexus, scaleni, recti, platysma, omohyoid

Course.—Chronic Remissions, but permanent recovery rare Life not shortened

Diagnosis.—

CLONIC TYPE—Simple, except from hysterical spasms

TONIC TYPE—From abnormal positions of head—

CONGENITAL TORTICOLLIS—*See below*

CERVICAL CARIES—Other signs present

HYSTERICAL SPASM

Transient—

FIBROSITIS, myositis. 'stiff neck'.

INFLAMMATION, e g, enlarged lymphatic glands or deep suppuration pyrexia and other signs

Treatment.—As in other 'tics' Remove local irritation Mental rest. Massage Movements of head Suggestion. Sedatives (avoid morphia)

OPERATION—Best is resection of part of spinal accessory, together with division of posterior primary divisions of 4 or 5 upper cervical nerves on other side Benefit often transient

CONGENITAL TORTICOLLIS.*

Origin from birth, often unnoticed for several years Probably due to congenital defect of centres in medulla; akin to congenital talipes

Characteristics.—(1) *Head rotated to other side and chin raised* sternomastoid shortened, hard and atrophied usually right side

(2) *Facial asymmetry*

Diagnosis.—Rupture of sternomastoid at birth also produces contraction, but thickening palpable at site of rupture

Treatment.—Tenotomy relieves torticollis facial asymmetry permanent

* Considered here for convenience

Section XII.—VASOMOTOR AND TROPHIC DISTURBANCES.

CHAPTER CXXXVI.

VASOMOTOR AND TROPHIC DISTURBANCES: TROPHONEUROSES.

I. RAYNAUD'S DISEASE.

A condition characterized by recurrent attacks of vascular spasm producing local syncope, terminating in gangrene in severe forms; usually affecting extremities, and generally bilateral and symmetrical. Probably due to a constitutional abnormality of the vasomotor mechanism.

Etiology.—

AGE.—First attack commonest in early adult life. No age exempt.

SEX.—Commoner in females.

HEREDITY.—Definite factor.

EXCITING CAUSE.—Cold is essential factor. Never occurs in warm climates.

PREDISPOSING FACTORS.—Gastric and intestinal disturbances may precede attack. Syphilis, malaria, and neuroses occasionally recorded.

Morbid Anatomy.—No constant changes. *Peripheral neuritis* not uncommon, but may be absent in typical and severe instances. Vessels, spinal cord, and brain usually normal.

Pathogenesis.—The phenomena undoubtedly result from spasm of arteries and arterioles, probably also of veins: has been observed in the retina. Slight cold produces results in liable subjects resembling effects of intense cold on healthy persons. Origin is a disturbance of the vasomotor innervation, which is abnormally sensitive to cold. No sufficient evidence to locate site of abnormality, whether in (1) vasomotor nerve fibres in vessels and peripheral nerves, or (2) vasomotor centres in cord and brain. Cord and brain usually normal; peripheral neuritis when present may be sequel of vascular changes.

Relation to Other Conditions.—Raynaud's disease is one of the 'trophoneuroses', a group of conditions which may be ascribed to abnormality of the vasomotor mechanism. The group includes many rare and obscure conditions, the separation and classification of which are still very doubtful.

Raynaud's disease has sometimes been closely grouped with paroxysmal hæmoglobinuria, erythromelalgia, and angioneurotic œdema, it being claimed that these tend to co-exist, or occur, in the same individual. With regard to this, note:—

Raynaud's Disease—Relation to other Conditions.

1. RAYNAUD'S DISEASE AND PAROXYSMAL HÆMOGLOBINURIA.—Attacks of latter common in Raynaud's disease. Relationship undeniable. Proves that Raynaud's disease is a widespread abnormality and not a local condition confined to the extremities.
2. RELATION TO ERYTHROMELALGIA.—Similarity occurs: (*a*) In hyperæmic stage of attack in Raynaud's disease, area being hot, throbbing, and vessels distended. (*b*) In later stages of chronic erythromelalgia, part may become blue and cold, rarely gangrene occurs. Differences are—
   RAYNAUD'S DISEASE.—(1) Commoner in females; (2) Cold is exciting cause; (3) Tends to be symmetrical and bilateral; (4) Area blue and cold; (5) Often paroxysmal.
   ERYTHROMELALGIA.—(1) Commoner in males, (2) Fatigue or heat excites onset. (3) Usually unilateral; (4) Area red and hot; (5) Often persists for years.
3. RELATION TO ANGIONEUROTIC ŒDEMA.—This disease is connected with urticaria and with group of conditions in which protein hypersensitiveness is a factor, e.g., bronchial asthma. There is no evidence that Raynaud's disease is related to these.

SUMMARY.—Association with paroxysmal hæmoglobinuria undoubted. Evidence does not yet definitely connect erythromelalgia more closely than as diseases of similar tissues. The conditions described in this chapter probably fall into at least two divisions: (1) Vasomotor disturbances, e.g., Raynaud's disease, paroxysmal hæmoglobinuria; (2) Connected with anaphylactic phenomena, e.g., angioneurotic œdema. Position of many diseases is doubtful, e.g., Milroy's disease, sclerodermia, thrombo angiitis deformans, facial hemiatrophy, intermittent hydrarthrosis (*See also* BRONCHIAL ASTHMA.)

Symptoms.—

GENERAL CHARACTERS.—(1) Affects extremities (circulation lowest). (2) Resembles results of extreme cold. (3) Tends to be bilateral and symmetrical. Very rare except in winter. Recurrences common, may be yearly. Ill health, gastric or intestinal disturbances, may precede attack.

SITES AFFECTED.—These are, in common order: (1) *Upper extremity.* Fingers first, especially index, rarely extends to wrist. Occasionally, areas on forearm. (2) *Lower extremity.* Toes first; rarely above ankles. (3) *Ears.* (4) *Nose.* Rarely, tongue, nates.

STAGES.—

1. LOCAL SYNCOPE.—From vasoconstriction (spasm) of arteries and arterioles, no blood enters area, which becomes white ('dead fingers'). Feeling of numbness, some stiffness and impairment of sensation. Returns to normal through asphyxia and hyperæmia. Duration, few minutes to hours.
2. LOCAL ASPHYXIA.—Colour of area blue to almost black. May follow stage of syncope, but in severe forms often

blue from onset. Ascribed to blood from veins flowing back into area before relaxation of arterioles. Affected area extremely cold, tender, and excessively painful.

3. ACTIVE HYPERÆMIA.—Arteries and arterioles dilate widely. Area red, hot, swollen, throbbing, and painful. Gradual return to normal.

4. GANGRENE.—If previous stages, with local cessation of circulation, be sufficiently severe and prolonged, natural sequence is necrosis of tissue, i.e., gangrene. Area becomes black, very cold, and very painful. Small bullæ with blood-stained fluid common. Gangrene usually (i) bilateral and symmetrical, (ii) dry, (iii) final loss of tissue small and usually superficial, e.g., end of one finger.

DEGREES OF SEVERITY.—

1. MILD ATTACKS.—Acrocyanosis, from spasm, followed by stages of asphyxia and hyperæmia and return to normal. All stages, white, blue, and red, often simultaneously present in different fingers or areas of one extremity, also patches of œdema. 'Chilblains' form a mild type.

2. MODERATE ATTACKS.—Area becomes permanently blue, in asphyxia; then gangrene follows and loss of tissue, e.g., tip of a finger. Pain extreme.

3. SEVERE ATTACKS.—Large area affected, e.g., both hands and both feet. Attacks often recurrent, and final loss of tissue extensive. Rare.

Complications.—Generally referable to vascular spasm, or to vasomotor phenomena

1. PAROXYSMAL HÆMOGLOBINURIA. *See above*, and also p. 559.

Following are all rare:—

2. CEREBRAL SYMPTOMS.—Transient aphasia; transient hemiplegia; epileptic fits.

3. TEMPORARY AMBLYOPIA.—From spasm of retinal vessels.

4. SKIN.—Urticaria. Rarely sclerodermia.

5. ARTHRITIS.—Effusion into joints. Occasionally fibrous ankylosis.

Albuminuria occasionally.

Diagnosis.—Usually simple.

GANGRENE of extremities occurs also in: senile gangrene, diabetes, advanced arteriosclerosis, and in the rare condition obliterative arteritis.

MULTIPLE AREAS OF GANGRENE rarely follow acute fevers, e.g., typhus, typhoid, malaria.

Treatment.—

PROPHYLAXIS.—Warm clothes. Sufficiency of fat in diet, and tonics. Careful attention to digestion and bowels. Avoid washing cold hands in hot water. Wintering in warm climates usually a complete preventive.

Soak extremities night and morning in water at 98° to 99° for ten minutes.

If Wassermann reaction positive, usual antisyphilitic treatment.

Raynaud's Disease—Treatment, *continued.*

DURING ATTACKS.—Wrap part affected in cotton-wool. Protect from injury. Pain may need morphia.

DRUGS.—Beneficial action doubtful. Calcium lactate, gr. xxx daily for three days, then omit. Nitroglycerin.

✓ TOURNIQUET applied until extremity becomes bright red (a few minutes), and repeated frequently (Cushing).

II. ERYTHROMELALGIA.

(*Red Neuralgia.*)

"A chronic disease in which a part or parts—usually one or more extremities—suffer with pain, flushing, and local fever, made far worse if the parts hang down" (Weir Mitchell). Rare disease.

Etiology.—

AGE.—Begins usually in middle age, or later.

SEX.—Commoner in males.

EXCITING CAUSES.—Fatigue, and hanging down limb. Hot weather. No hereditary factor apparent. Local injury may precede onset.

Morbid Anatomy.—Small arteries and veins are thickened. No evidence of peripheral neuritis.

Pathogenesis.—Obscure. May be abnormality of vasomotor centres or of blood-vessels themselves.

Note.—

(1) In arsenical neuritis, similar condition may occur. Rarely in syringomyelia, myelitis, tabes, disseminated sclerosis.

(2) In intermittent claudication, in which vascular thickening is present, condition resembling erythromelalgia may occur and gangrene follow.

(3) Relation to Raynaud's disease, referred to above.

Symptoms.—

SITE.—Most commonly one foot: rarely extends above ankle. Occasionally bilateral. Rarely hands and face.

ONSET.—In hot weather. Burning pain in sole after walking, recovers on rest; then with recurrent attacks affected area becomes red, hot, slightly swollen; arteries throbbing, veins enlarged; pain extreme. Surface temperature higher than on unaffected areas. No pitting.

Condition eased by elevation or cold: aggravated by hanging down or heat.

DURATION.—Few hours to weeks. *Chronic condition* may develop. Part later may become blue and cold, and gangrene follow.

Treatment.—At onset, prolonged rest and elevation for many weeks. Cold climate advisable. When condition developed, cold applications and elevation; pain may necessitate morphia. Amputation of doubtful value.

III. ANGIONEUROTIC ŒDEMA.

A condition characterized by the sudden occurrence of œdematous swellings of local extent and of short duration.

Etiology.—Occurs at any age and in either sex. *Heredity* frequent; through many generations. Neurotic factor in some forms.

Pathogenesis.—Is connected with urticaria and a group of conditions in which protein hypersensitiveness is a factor (*see* RAYNAUD'S DISEASE and BRONCHIAL ASTHMA).

Symptoms.—

ONSET.—Sudden Occasionally preceded by local itching and heat.
CHARACTERS.—Local œdematous swelling: firm, rarely pits, definite outline.
SITE.—Hands, face, feet, and genitals commonest.
DURATION OF SWELLING.—Transient, often few hours; rapid disappearance. Frequently recurs in a different site.
GENERAL SYMPTOMS.—Occasionally gastro-intestinal disturbances, e.g., colic.
ŒDEMA OF LARYNX.—Often fatal. Constitutes essential danger.

Prognosis.—Œdema of the larynx often rapidly fatal (in absence of tracheotomy). Otherwise swellings are but a passing inconvenience.

Treatment.—General health. Nitroglycerin and calcium lactate should be tried.

IV. HEREDITARY ŒDEMA OF THE LEGS.

(*Milroy's Disease. Chronic Trophœdema.*)

A condition characterized by chronic œdema of the legs without obvious cause. Probably due to increased permeability of vessels, a trophoneurosis.

Chief Characters.—

1. Hereditary and familial disease. Sexes equal. Often from birth.
2. No obvious cause.
3. Chronicity. Condition permanent.
4. Swelling usually of lower extremities, pits on pressure, finally great hypertrophy. Swelling circumscribed and foot may escape.

Acute periods occur with fever and increased swelling, possibly analogous to inflammatory attacks in elephantiasis.

V. FACIAL HEMIATROPHY.

A rare condition of unknown origin, characterized by slow progressive unilateral wasting of the tissues of the face, the muscles being least affected.

Etiology.—*Onset* in childhood: rarely in adults. Females commonest. *Predisposing factors*: slight injuries, acute infectious fevers: may be none. *Heredity* slight.

Facial Hemiatrophy, *continued.*

Pathogenesis.—Probably connected with 5th nerve, developmental or possibly morphœa. May be extreme neuritis of 5th nerve.

Characteristics.—(1) Strictly unilateral. (2) Onset insidious and progress slow. (3) Commences in an area or areas of skin with local wasting. (4) Extends gradually: involves fat and subcutaneous tissue until entire side of face affected. (5) Hair may whiten or fall out. (6) Bones: growth retarded, or atrophy and teeth fall out. (7) Tongue: may be hemiatrophy. (8) Facial muscles: little or no affection, except loss of fat. No sensory or electrical changes: may be slight tingling. Rarely: bilateral, or extends to upper limb. *Course*: progressive to a certain stage and then stationary: no effect on life.

Diagnosis.—From: (1) Congenital torticollis with asymmetry; (2) Localized sclerodermia. Also from atrophy of hemiplegia, acute poliomyelitis, nuclear lesions.

Treatment.—Massage and electrical treatment. Paraffin injections under skin.

Section XIII.—DISEASES OF THE MUSCLES, JOINTS, AND BONES.

CHAPTER CXXXVII.

DISEASES OF THE MUSCLES.

I. MYOSITIS.

Inflammation of muscles, usually confined to voluntary muscles.

Classification.*—

PRIMARY.—

1. SUPPURATIVE MYOSITIS.—Very rare.
2. DERMATOMYOSITIS.—Very rare.

SECONDARY.—

1. SUPPURATIVE MYOSITIS.—In pyæmia, etc.
2. TRICHINELLA SPIRALIS.
3. ACUTE SPECIFIC FEVERS.—Mainly degeneration, e.g., Zenker's degeneration, most common in enteric.

Rarely—

4. Syphilitic.
5. Tuberculous.

SPECIAL CHRONIC DISEASES.

1. MYOSITIS OSSIFICANS.—(*a*) Local. (*b*) General and progressive.
2. MYOSITIS FIBROSA.

FIBROSITIS AND MYOSITIS.—*See* p. 924.

PRIMARY SUPPURATIVE MYOSITIS.

Mainly recorded in Japan. Sudden onset, constitutional disturbances, muscles swollen and tender, with subsequent abscess formation. Various pyogenic organisms isolated.

DERMATOMYOSITIS.

Onset gradual or sudden, with constitutional disturbances: pyrexia and enlarged spleen.

Characteristics.—(1) *Muscles* swollen and tender: few escape. No abscess formation. (2) *Dermatitis* of various types, œdema, urticaria, purpura, erythematous or erysipelatous eruptions.

Morbid Anatomy.—Parenchymatous and interstitial inflammation of muscle.

Pathogenesis.—Allied to urticaria, purpuric, and similar conditions.

* Modified from Batten's article in Allbutt and Rolleston's *System of Medicine.*

Myositis, *continued.*

Diagnosis.—From trichiniasis, only by removal of portion of muscle, or possibly by X rays.

Prognosis.—Usually fatal, from respiratory disturbances.

Clinical Varieties.—A hæmorrhagic form occurs. Neuromyositis: sensory changes described.

MYOSITIS OSSIFICANS PROGRESSIVA.

A generalized, irregular, progressive ossification of voluntary muscles. Distinguished from ossification of a single muscle, e.g., 'rider's bone'.

Etiology.—*Onset* in early infancy. *Males* commonest. *Microdactyly* of thumb and great toe common. Pathogenesis unknown.

Symptoms.—Commences in muscles of back and neck. *Four stages*, proceeding simultaneously in different sites: (1) Acute attacks of pain and swelling of muscles, subsiding in few weeks. (2) Attacks recur and fibrosis follows, forming local tumours. (3) Ossification develops in tumours after further attacks, irregular masses gradually coalescing into shapes 'like coral': still movable on deep tissues. (4) Bony masses become adherent to bones, producing absolute immobility. Aponeuroses, tendons, joints, etc., become affected. Few or no voluntary muscles unaffected.

Course.—Progresses by recurrence of acute attacks. Finally, after years, unable to move or masticate. Death from intercurrent diseases.

Diagnosis.—Early stages: from injury or rheumatism. Later stages: from congenital multiple exostoses.

Treatment.—Palliative.

MYOSITIS FIBROSA.

Very rare. Fibrosis of muscles, commencing in early life, usually in lower extremities, progressing gradually to contractures and immobility: no ossification occurs: joints unaffected.

Pathology.—Great increase of fibrous tissue.

Diagnosis.—From acute arthritis of children, myopathies, and cerebral diplegias.

Treatment.—Massage, movement, and electricity: recovery under treatment is recorded.

II. FIBROSITIS.

(*Myalgia. Myositis. Lumbago and other types.*)

A painful condition of various voluntary muscles, due to inflammation of the insertions, fibrous sheaths, and periosteal attachments.

Etiology.—(1) *Indirect trauma*, by strain or sudden severe muscular contractions. (2) *Exposure* to wet, cold, or draughts of air. *Gout* is a predisposing factor. *Chronic forms* occur in later life.

Pathology.—Inflammation of, and later, proliferation of, the fibrous tissue of the muscle sheaths, insertions, ligaments, and periosteum. Fibroid nodules may form.

Symptoms.—

ONSET sudden. Various local sites attacked in different types. Constitutional symptoms absent or very slight. Pain severe: acute severe spasm on contracting affected muscles, especially suddenly: may be dull ache in interval. Muscles often tender; may be indurated, especially in neck.

DURATION.—Few days to weeks.

RECURRENCES.—Very common.

Types.

LUMBAGO.—Affects lumbar muscles. *Onset* usually absolutely sudden, with or without causal strain. *Pain* extreme on contracting back muscles, e.g., on regaining erect position after stooping. Patient walks slowly with rigid back. Recurrence very common.

Diagnosis, in recurrent or persistent attacks, from: sacro-iliac disease, caries, arthritis, or rarely spinal tumours.

PLEURODYNIA.—Affects intercostal muscles: unilateral. *Pain* extreme.

Diagnosis from pleurisy, intercostal neuralgia (no tenderness of nerve trunks).

STIFF NECK: ACUTE TORTICOLLIS.—Very common in children, following draughts or strained position of neck. Muscles tender and often indurated.

Treatment.—

REST.—Avoid muscular contractions. Local rest assisted by strapping.

OPEN BOWELS FREELY. Light diet. Much bland fluid.

DRUGS.—Salicylates, aspirin, colchicum, guaiacum, and iodides, prescribed as in gout (*see* p. 313).

LOCAL TREATMENT.—

HEAT.—Hot sand-bag. Poultices.

COUNTER-IRRITANTS.—Blisters. Cautery. Tincture of iodine. Liniment of aconite, belladonna, and chloroform.

ACUPUNCTURE.—Sterilize skin. Plunge sterilized needles about 3 inches deep, leave five to ten minutes. Often extremely effective.

CHRONIC CASES.—Hot-air baths, diathermy, cataphoresis, light massage, and finally spa treatment may be effective. Avoid morphia for pain.

CHAPTER CXXXVIII.

ARTHRITIS DEFORMANS.

(*Rheumatoid Arthritis. Osteo-arthritis.*)

A disease of the joints of unknown origin, characterized by changes of various degrees in the synovial membranes, peri-articular tissues, cartilage, and bones, resulting in pain, limitation of movement, muscular wasting, and deformities.

CLASSIFICATION OF TYPES.

Two main groups are distinguishable :—

1. PERI-ARTICULAR TYPE (Rheumatoid Arthritis).—Characteristically acute. (i) Age at onset 20 to 40 years ; (ii) Onset usually acute ; (iii) Peri-articular tissues mainly affected ; (iv) Joints have *fusiform* shape. Mild septic foci not uncommon. Rarely, enlargement of lymphatic glands and, it is said, of spleen.
2. OSTEO-ARTHRITIC TYPE (Osteo-arthritis).—Characteristically chronic. (i) Age at onset 40 to 60 years ; (ii) Onset chronic ; (iii) Injury and exposure not uncommon ; (iv) Cartilage and bone mainly affected ; (v) Joints have *nodular* shape.

There is not yet agreement as to whether these types are separate entities or manifestations of the same disease. Intermediate forms admittedly exist ; moreover, the late stages of the peri-articular type, after many years, may resemble closely the osteo-arthritic type. But the differences in clinical course and pathological changes are sufficient to demand separate descriptions.

ATROPHIC TYPE.—This is a third group described by some authorities, and by others regarded as a late stage of the peri-articular type, the earlier stage being 'exudative' : of this type it is almost an inevitable sequel, but in rare instances it may be primary.

Other groups which have been distinguished include :—

STILL'S DISEASE.—Suggests peri-articular type occurring in childhood.

MONO-ARTICULAR ARTHRITIS.

SPONDYLITIS DEFORMANS.—These last two types are special forms of the osteo-arthritic group.

There is no relation to gout or acute rheumatic fever. That the disease is a septic infection or is due to the action of bacterial toxins has been claimed, especially for peri-articular type. Evidence advanced : (1) Acute onset, often with definite pyrexia ; (2) General resemblance to septic joints ; (3) Septic foci common ; (4) Occasional enlargement of lymphatic glands. No

unimpeachable organism has been described. Other theories include a gastro-intestinal toxæmia, e.g., 'intestinal stasis'.

Note.—The division into two main groups (which is here adopted) is not universally accepted, and the nomenclature is not yet settled even by those who follow it. Thus 'arthritis deformans', 'rheumatoid arthritis', and 'osteo-arthritis' are still used by many as synonymous terms.

MORBID ANATOMY.*

1. ACUTE OR PERI-ARTICULAR TYPE (Rheumatoid Arthritis). – (i) *Early 'exudative' stage*: Thickening of synovial membrane and peri-articular tissues: main cause of enlargement of joint. Effusion variable. Red, vascular, villous outgrowths of synovial membrane. Bone and cartilage little changed, but signs of thinning from pressure of synovial membrane. (ii) *Late 'atrophic' stage*: Thickened synovial membrane atrophies and fibroses. Cartilage and articular surfaces destroyed, commencing at site of pressure of synovial membrane. Bone in neighbourhood rarefies. Fibrous adhesions between surfaces; may ossify. Proliferation of bone slight, may be a few spicules, no definite osteophytes.

2. CHRONIC OR OSTEO-ARTHRITIC TYPE.—Earliest changes occur in cartilage, viz., fibrillation and erosion.

   CARTILAGE cells proliferate, capsules burst into joint; ground substance thus divided into filaments and devoid of cells. Cartilage has velvety appearance, wears away, and exposes bone.

   BONE, exposed, hardens on surface and has ivory appearance (eburnation), grooves form from movements of surfaces and cause 'crepitus'. Atrophy of bone variable, may be considerable, e.g., in senile hip-joints. '*Osteophytes*' form at edges, proliferation of cartilage cells producing accumulations which ossify, i.e., 'lipping' of joint, and, by interlocking, render joint immobile. Hypertrophy of bone also occurs. True bony ankylosis very rare, except in spine, where bone may also form in ligaments.

   SYNOVIAL MEMBRANE.—Thickens. Fringes may hypertrophy, become cartilaginous, separate, and constitute 'foreign bodies' in joint.

MUSCULAR ATROPHY.—Rarely absent. Contractures occur.

Other lesions include: Trophic changes in skin, Heberden's nodes, and rarely subcutaneous fibroid nodules. Visceral lesions are rare, most frequent being pleurisy.

METABOLISM.—Increased excretion of organic phosphates, retention of calcium, magnesium, and phosphorus. Slight acidosis. No increase of ethereal sulphates.

Radiographs exhibit well the changes as described.

* *See* Garrod in Allbutt and Rolleston's *System of Medicine.*

Arthritis Deformans, *continued.*

✓ ACUTE OR PERI-ARTICULAR TYPE.

(*Rheumatoid Arthritis.*)

Etiology.—Age at onset, 20 to 40 years. General physique usually poor. Females predominate. Mild septic foci may be present, especially pyorrhœa alveolaris and vaginal discharge, but often none found. May follow repeated pregnancies or the menopause.

Symptoms in Early 'Exudative' Stage.—

ONSET.—Usually acute or subacute. Generally many joints.

PAIN.—Variable, often severe. Slight at rest but severe on movement. Worse at night. Partly due to muscular spasm.

CONDITION OF JOINTS.—*Fusiform* swelling, due to swollen joint and wasted muscles. Skin appears sodden, but little redness. Swelling mainly of peri-articular tissues: may be some synovial effusion.

JOINTS AFFECTED.—Order of frequency: (1) Hands and feet; proximal interphalangeal and metacarpophalangeal joints. (2) Wrists. (3) Ankles. (4) Knees. Temporomaxillary joint and cervical vertebræ also very common. No joint immune.

TEMPERATURE.—In acute onset, occasionally 102° to 103°, subsides to 100°, and may persist for weeks. Often much slighter. Pulse in proportion to temperature.

LIMITATION OF MOVEMENT.

MUSCULAR WASTING AND CONTRACTURES.—Early and rapid.

LYMPHATIC GLANDS may enlarge, and perhaps the spleen.

Progress.—Often prolonged. Various joints frequently attacked in succession. Fine crepitus develops in joints.

Symptoms in Late 'Atrophic' Stage.—Swelling diminishes and becomes less fusiform. Muscular wasting extreme and contractures marked. Subluxation of joints common. Results in severe deformity, fixation and loss of function in joints. Trophic changes in skin and nails. Pain may subside, but spasms in limbs often troublesome. Rarely this syndrome occurs as primary type.

Diagnosis.—

1. RHEUMATIC FEVER.—Often very difficult. In rheumatoid arthritis: (i) Little or no response to salicylates; (ii) Smaller joints commoner, pain and tenderness rarely very severe; (iii) Does not subside in one joint when commencing in another; (iv) Temporomaxillary joint and neck often affected; (v) No endocarditis; (vi) Subsequent joint changes.
2. GONORRHŒA.—Very difficult. In gonorrhœa: (i) History and presence of gonococci; (ii) Small joints less common; (iii) Often wanders from one joint to another, but specially injures one. Joint hot and œdematous.

3. GOUT.—In gout: (i) Commoner in men; (ii) Onset sudden; (iii) Great toe and thumb especially; (iv) Joint 'swollen, red, shiny, and œdematous'; (v) Pain severe. In chronic gout, more difficult; usually previous acute attacks.
4. TENOSYNOVITIS.—Creaking over tendon, joint change slight, pain increased on movement.
5. CHARCOT'S JOINT.—Sudden painless swelling, much effusion, evidence of syphilis.

Radiograph.—Especially: (1) Rarefaction of bone near joints; (2) Proximity of joint surfaces owing to destruction of cartilages. No lipping or osteophytes.

CHRONIC OR OSTEO-ARTHRITIC TYPE.

Etiology.—Age of onset 40 to 60 years. In general forms females predominate; in spondylitis and mono-articular forms, males commoner.

PREDISPOSING FACTORS.—Injury, exposure to cold and wet, general ill-health. *Pyorrhœa alveolaris, carious or deficient teeth,* practically never absent.

Symptoms.—

ONSET.—Chronic, rarely acute. Generally polyarticular. Exacerbations and gradual progress usual.

PAIN.—Variable. May be slight throughout. Sometimes severe.

CONDITION OF JOINTS.—Swelling tends to be nodular in shape, nearly confined to joint, and affection of peri-articular structures slight.

JOINTS AFFECTED.—Distribution may be: (1) Polyarticular, either from onset or by subsequent extension: usually a few large joints, but no joint immune. (2) Mono-articular, especially vertebræ (spondylitis), hip-joint, and knee.

Constitutional symptoms slight. Temperature slight. No enlarged glands.

Advance and exacerbations occur until final development of characteristic condition.

Characteristics in Late Stages.—

1. PAIN.—Often in wet weather, but also when hot and dry. Worse at night.
2. DEFORMITY OF JOINTS.—Due to: (i) Thickening of capsule. (ii) Osteophytes and overgrowth of bone ('lipping'). (iii) Absorption of cartilage and bone altering shape of joint-surfaces and angles of articulation: subluxation may occur. (iv) Muscular contractures. '*Ulnar deviation*' characteristic: due to affection of metacarpophalangeal joints.
3. LIMITATION OF MOVEMENT.—From locking of osteophytes, fibrous adhesions, and causes of deformity. Bony ankylosis very rare except in spine.
4. MUSCULAR WASTING.—Constant, but not extreme. Reflexes are increased. Cause doubtful.

Arthritis Deformans—Chronic or Osteo-arthritic type, *continued.*

5. CREPITUS ON MOVEMENT.—Fine in early stage; coarse later. From apposition of bony surfaces and formation of grooves.

SKIN.—Often glossy. Trophic changes in nails. Occasionally pigmentation.

PALLOR usual, and some anæmia.

Final Condition.—Patient may become helpless, at which stage condition often quiescent and painless. Not infrequently small joints of hands escape when large joints are severely affected, and vice versa.

Heberden's Nodes.—Small bony swellings, usually on distal side of terminal interphalangeal joints: apparently from tubercles on insertion of extensor tendons. Commoner in women. May be first sign of arthritis. Similar bone swellings occur in gout, though rarely.

Diagnosis.—Usually simple.

HIP-JOINT.—From tuberculosis (rotation specially affected) and sacro-iliac disease.

SPONDYLITIS.—From tuberculous or pressure caries (compression myelitis).

SHOULDER-JOINT.—From neuritis and subdeltoid bursitis.

Radiograph every doubtful case.

Radiograph.—Especially lipping of margins and osteophytes.

MONO-ARTICULAR TYPE.

Common sites:—

Hip-Joint.—

ETIOLOGY.—Old age, especially males. Injury common cause. One joint, or less commonly both joints, affected.

SYMPTOMS.—(1) *Pain*, severe in groin and front of thigh, often referred to knee. (2) Limitation of movement. (3) Muscular wasting, especially thigh and buttock. (4) True shortening may occur from marked atrophy of bone surface. Often much 'lipping'. Rarely: Baker's cysts.

Knee.—Commoner in women, often at menopause.

SYMPTOMS.—(1) *Pain*; (2) *Crepitus*; (3) Deformities and limitation of movement. Lipping common. 'Foreign bodies' not infrequent.

Shoulder.—Common in both sexes.

Thumb (metacarpophalangeal joint).—Also common.

SPONDYLITIS DEFORMANS.

Applied to forms of arthritis deformans in which the spine is chiefly, widely, and severely affected.

General Characters.—(1) *Males* commoner. (2) Injury apparently a factor. (3) *Entire spine* usually affected: severest in upper

dorsal and cervical regions. (4) *Bony ankylosis* of intervertebral joints, and sometimes ossification of spinal ligaments. (5) *Immobility of spine*; also of thorax from ankylosis of joints of ribs and spine, whence fixation of ribs and abdominal breathing. (6) *Nerve-root pressure symptoms.*

Final condition may be: (*a*) Straight 'poker-back', (*b*) Bent back with lordosis, common in agricultural labourers.

Groups.—Two groups are described:—

1. VON BECHTEREW.—(i) Spine alone affected. (ii) Nerve-root pressure symptoms marked, e.g., pain, paræsthesia, muscular atrophy. Von Bechterew held spinal meningitis to be the initial lesion.

STRUMPELL-MARIE'S 'SPONDYLOSE RHIZOMÉLIQUE'.—(i) Hip and shoulder-joints also affected. (ii) Nerve symptoms less marked.

[illegible] ypsy, evidence of pressure on nerve roots is rarely marked. Diff[illegible]tion of the two groups is indefinite, and both are pro[illegible]rthritis deformans.

Diagnosis.—*Gonorrhœa* also may affect spine, and produce rigidity.

Note.—Vertebræ are also affected in ordinary types: (*a*) In acute type, especially cervical; complete recovery usual. (*b*) In chronic type, especially lower dorsal and lumbar; rigidity common, sciatica and referred pains occur.

PROGNOSIS OF ARTHRITIS DEFORMANS.

Prognosis in general is bad; recurrence and advance are usual. Questions arising are: (1) Will the disease be arrested? (2) What deformity and limitation of movement will result? Most important data are general nutrition of patient in the first question, and changes in bone and cartilage as shown by X rays in the second.

GENERAL FACTORS IN PROGNOSIS.—

1. Early diagnosis.
2. Social position and occupation. Permitting long treatment
3. General condition of nutrition. Thin 'dried-up' patients are difficult.
4. Discovery of septic foci. Treatment improves prognosis.
5. Acute forms better than chronic, i.e., 'peri-articular' better than 'osteo-arthritic'.
6. X rays. Changes in bone and cartilage are serious.
7. Rapid progress and attack on joints consecutively is bad, but a severe initial attack may subside.
8. Pain may hinder massage and movement, thus affecting treatment.

ACUTE OR PERI-ARTICULAR TYPE.—Duration of temperature and of joint swellings is a guide. Bad prognosis in (*a*) rapid muscular atrophy and contractures, (*b*) onset after menopause.

CHRONIC OR OSTEO-ARTHRITIC TYPE.—General prognosis poor.

Arthritis Deformans—Prognosis, *continued.*

SPECIAL MANIFESTATIONS.—

MONO-ARTICULAR TYPE (e.g., hip, shoulder).—Often remains localized, though progress usual in affected joint.

SPONDYLITIS DEFORMANS.—Prognosis bad, fixation of spine usual. Avoidance of trauma important.

RHEUMATOID ARTHRITIS IN CHILDREN.—Prognosis very bad.

TEMPOROMAXILLARY AND CERVICAL VERTEBRAL JOINTS.—Recovery usual (possibly from constant movement in eating and talking).

TREATMENT.

Early diagnosis and *patience* are first essentials. *Indications*: (1) Remove septic foci; (2) Treat general health; (3) Treat joints.

SEPTIC FOCI.—Especially *teeth*, also tonsils, and genito-urinary system.

GENERAL HEALTH.—

1. DIET.—Full diet, liberal fats and proteins. Alcohol permissible. Correct gastric disturbances.
2. REST IN BED.—During pyrexia only: always massage.
3. DRUGS.—Tonics, especially syrupus ferri iodidi (ʒj t.d.s.).

CLIMATE.—Dry and sunny: on sand or gravel. Avoid clay and seaside. Dry moorland good. Egypt, Algiers best.

CLOTHING.—Sufficiently warm.

JOINTS.—Rest in good position during acute pyrexial stage.

1. MOVEMENTS AND EXERCISE.—Of highest importance, and needing patience and perseverance; for prevention of fixation, contractures, and muscle wasting.
2. MASSAGE.—In acute stages: must be light.
3. COUNTER-IRRITANTS.—Blisters. Painting with iodine.
4. DRUGS.—Either:—
   a. Guaiacum and iodine.—Various methods, e.g.: (i) Guaiacol carbonate gr. v to x, t.d.s., in cachets, and potassium iodide gr. v to x, t.d.s., in a mixture; (ii) Tinct. guaiaci ammoniata ʒss, and potassium iodide gr. v to x, t.d.s., in a mixture (tincture must be fresh, and is unpleasant). Or:—
   b. Sodium salicylate or aspirin.

PAIN.—Scott's dressing or paint with methyl salicylate. Aspirin.

SPONDYLITIS.—Protective jacket. Avoid injury.

SPECIAL METHODS FOR CHRONIC STAGES.—

1. HYDROTHERAPEUTICS AND SPA TREATMENT.—Radio-active and peat waters best. Much idiosyncrasy, but results often good. Spas: Bath, Buxton, Harrogate, Strathpeffer (peat), Woodhall Spa; Aix-les-Bains, Mont Dore.
2. RADIANT HEAT AND HOT-AIR BATHS (e.g., Dowsing and Tyrnauer methods).—Relieve pain, but often temporarily.
3. ELECTRICAL TREATMENT AND CATAPHORESIS (sodium salicylate).

VACCINE TREATMENT.—Isolation of organism for autogenous vaccine. Results doubtful.

GENERAL SUMMARY.—Early diagnosis, patience in treatment, maintenance of general health, removal of sepsis, prevention of deformities by constant exercise. (The teeth and mouth should be attended to before other treatment is commenced.)

ARTHRITIS DEFORMANS IN CHILDREN.

(*Still's Disease.*)

General Characters.—

ONSET.—Insidious usually: less often acute. Age 3 to 6 years at onset.

JOINTS.—Enlarged. Swelling 'fusiform', mainly of peri-articular tissues, characters resembling acute peri-articular type. Muscular wasting severe, and limitation of movement.

LYMPHATIC GLANDS.—Enlarged. Generalized enlargement, usually of considerable size: may increase during exacerbations.

SPLEEN.—Often palpable.

TEMPERATURE.—Often persistently about 100°. Sweating common.

PROGRESS.—Slow advance, with exacerbations and pyrexia. Anæmia, wasting, debility, and lack of development. Heart unaffected. Intercurrent diseases often fatal.

Note.—Several rare and obscure groups of arthritis and joint changes occur in children. Their relation to and differentiation from Still's disease has been little studied. (*See* LATE RICKETS, p. 350.)

CHAPTER CXXXIX.

DISEASES OF THE BONES.

I. HYPERTROPHIC PULMONARY ARTHROPATHY.

A symmetrical enlargement of the bones of the extremities of the limbs, with 'clubbing' of the terminal phalanges. Associated with certain diseases, especially of the lungs: never primary. Very rare.

1. **'Clubbing of the Fingers'** (*Hippocratic fingers*).—An initial and allied condition. Very common.

DESCRIPTION.—Terminal phalanges swollen and rounded. Nails enlarged and curved in both directions. Skin shiny. No pain. Toes also affected occasionally: usually congenital morbus cordis. Onset usually gradual. Rarely in two weeks in empyema: may disappear after treatment.

ETIOLOGY.—

1. CONGENITAL MORBUS CORDIS.—Common. Very rare in acquired cardiac lesions.
2. DISEASES OF THE LUNGS.—(i) Bronchiectasis; (ii) Phthisis, especially with cavities; (iii) Empyema. Rarely in abscess

Clubbing of the Fingers, *continued.*

of lung, emphysema, etc. In aneurysm, rarely: sometimes unilateral.

3. Certain other conditions, rarely, e.g., congenital syphilis, chronic jaundice, chronic diarrhœa.

MORBID ANATOMY.—Thickening of fibrous tissues, and distention of vessels. No bony changes.

2. **Hypertrophic Pulmonary Arthropathy** (*Marie's syndrome*).—

DESCRIPTION.—(1) Hands and feet large. (2) Clubbing of terminal phalanges invariable. (3) Forearm thickened near wrist; to less degree long bones near ankle. Rarely: enlargement at knee and elbow-joint. Occasionally kyphosis. Condition symmetrical: less in lower extremities. Slight stiffness of joints. May be tenderness, but no redness or actual pain. Onset gradual, usually unnoticed by patient.

ETIOLOGY.—As in clubbed fingers, except that occurrence in morbus cordis is *extremely rare*. Commoner in males.

MORBID ANATOMY.—Proliferation of bone under periosteum (an ossifying periostitis), causing enlargement. Rarefaction of deeper bone tissue. Synovial membrane may thicken.

PATHOGENESIS.—Obscure. Allied condition of clubbing often ascribed to congestion, but does not explain non-thoracic conditions. Marie's theory: periostitis due to toxins: not improbable. Other theories include: tuberculous periostitis (many authorities); neuritis and œdema (now discarded).

SITES AFFECTED.—Usual are: lower ends of ulna, radius, tibia, and to less degree fibula, also metacarpals and metatarsals. Carpal and tarsal bones escape. Rarely, lower end of femur and humerus, and patella. Face never affected.

DIAGNOSIS.—Usually simple. Clubbing of fingers and primary disease invariably present. Radiograph shows bony changes. Skull never affected. Diagnosis, rarely difficult, from acromegaly, osteitis deformans, arthritis deformans. Condition does not influence prognosis as to life.

II. OSTEITIS DEFORMANS.

(*Paget's Disease.*)

A chronic disease of bones occurring in later years, producing softening, new formation, and subsequent hardening; and resulting especially in enlargement of the head, curving of the spine, and curving and enlargement of the bones of the legs. Rare disease.

Etiology.—*Age*: rarely under 50 years. Some evidence of heredity. No factors known, but arteriosclerosis invariable.

Morbid Anatomy.—Apparently a chronic inflammation, a rarefying osteitis. (1) Early stages: bones softer and become more vascular, hence *curvatures* from pressure. (2) Later: deposits of new bone both in medulla and also, markedly, under periosteum, mainly along normal ridges. (3) Finally, hardening of the bones.

BONES AFFECTED AND RESULTING CHANGES.—

1. SKULL.—Great enlargement. Thickness ½ to ¾ inch.
2. SPINE.—Kyphosis.
3. TIBIA.—Great thickening and bowing: convexity forwards.

Changes less marked in femur. Pelvis broadens. Clavicles, thick and deformed. Ribs fall in.

Face, hands, feet little change. Upper extremity less than lower.

Symptoms.—*Onset* insidious: often first noted by friends. General health good.

Early noticeable phenomena: (1) Head enlarging. (2) Bowing of legs. (3) Stature shortening (from kyphosis and curvature of legs).

Condition developed: Forehead prominent and face appears small (thus differing from acromegaly). Spine bent, and chin held forward. Legs bowed, with enlargement, often enormous, of tibiæ. Sometimes thickening of clavicles, thorax fallen in, and abdomen prominent.

Variations.—Occasionally is painful. Changes may be confined to tibia and fibula. *Osteosarcoma* not uncommon: also various bone tumours and cysts, occasionally numerous ('multiple hyperostoses', 'tumour-forming osteitis deformans').

III. LEONTIASIS OSSEA.

Hyperostoses of cranial and facial bones. Extent and distribution variable; occasionally superior maxillæ affected alone, or other bones of body affected also, but less severely. Formation of dense new bone results in. (1) Large, grossly deformed head. (2) Severe pains, blindness, deafness, etc., from obliteration of foramina, pressure on and destruction of nerves, and reduction in size of cavities, e.g., orbit and mouth.

Very rare. Onset about 30 years. Progress slow. Possibly a variety of osteitis deformans.

IV. OSTEOGENESIS IMPERFECTA.

(*Fragilitas Ossium. Osteopsathyrosis Congenita. Annular Rickets.*)

An intra-uterine defect characterized by abnormal brittleness of bones, due to failure of membrane and periosteal bone-formation, and resulting in numerous fractures.

Description.—Fœtus nearly always born dead. Characterized by: (1) Body proportions and length of bones fairly normal. (2) Bones brittle or soft, sometimes can be bent. (3) Numerous intra-uterine fractures; callosities at site of union. (4) Cranium development defective. (Some of the callosities are possibly abnormal bone-formation, and not results of fractures.)

Morbid Anatomy.—*See* OSTEOPSATHYROSIS, which is probably identical, the subjects surviving infancy.

V. OSTEOPSATHYROSIS.

(*Fragilitas Ossium. Lobstein's Disease.*)

A rare condition, probably of intra-uterine origin, characterized by abnormal brittleness of bones, due to failure of membrane and periosteal bone-formation, and resulting in frequent fractures.

Etiology.—Subjects are probably survivors of *osteogenesis imperfecta.* Origin unknown; possibly faulty internal secretion. No reaction to treatment for rickets or syphilis.

Morbid Anatomy.—Cartilage bone-formation unaffected, hence no shortening of bone, and body proportions normal. Subperiosteal and membrane bone-formation defective, consequently cortex is thin, and bones brittle and easily fractured. (Pathology is thus converse of achondroplasia, in which membrane bone-formation is normal, and endochondral ossification is defective.)

Symptoms.—General health unaffected. Fractures occur with extreme ease, and unite rapidly. Tendency present from birth, and usually ceases about 30 years of age. Subsequent life depends on deformities from repeated fractures.

Treatment.—No specific. Phosphorus. Protection against injury.

Note.—*Abnormal fragility of bones* also occurs in old age, insanity, various bone lesions (e.g., syphilis, sarcoma, and secondary tumours), cachectic conditions, rickets, scurvy, tabes, and phosphorus poisoning. Also with 'blue sclerotics': condition hereditary, connected with deficiency of calcium.

VI. ACHONDROPLASIA.

(*Chondrodystrophia Fœtalis.*)

An abnormality of cartilage bone-formation arising in fœtal life, resulting in deficient growth of long bones. Surviving subjects are dwarfs with short limbs and long bodies.

Description.—Main characters:—

1. DWARFS.—Height 3 to 4 feet.
2. EXTREMITIES VERY SHORT.—*Especially femur and humerus.* Fingers reach iliac crest.
3. TRUNK about normal.
4. HEAD.—Appears large. Face small with pug-nose. (Vault normal, base affected.)
5. 'TRIDENT HAND'.—Fingers of equal length and diverging.

Other features.—*Sacrum* tilted forward, whence: (1) Pelvis contracted; (2) Apparent lordosis, but spine actually very straight; (3) Abdomen prominent. *Limbs* bowed and bent owing to abnormal articulations and not to curving of bones. *Feet* large and flat, tissue round ankle in folds.

Acetabula set far back, hence nates prominent. Various congenital deformities, e.g., hypospadias not infrequent.

General features.—If surviving first year, general virility marked (normal heart in small body). Mental development normal or quaint. Muscles and bones very strong. Sexually precocious. Often gymnasts or public entertainers.

Morbid Anatomy.—Essential change is deficiency of *endochondral ossification* (cartilage bone-formation), due to abnormality of epiphyseal cartilages. Line of ossification is straight, but narrow (*see* RICKETS, p. 346). Zone of cartilage cell-proliferation shows characteristic changes:—

1. Cartilage cells irregularly arranged and *very scanty*, i.e., aplasia.
2. Connective-tissue strands grow in from periosteum, and may completely separate shaft from epiphysis.

Ossification of epiphyses either retarded or premature: may be early union to shaft.

Periosteal bone-formation normal.

Pathogenesis.—Is undetermined. Always commences in fœtal life, apparently between third and sixth month. Bones which are laid down in cartilage after this, and all membrane bones, escape (Symington and Thomson). Many born dead. Sexes equal.

THEORIES.—

FŒTAL RICKETS.—Cartilage changes differ from rickets, being aplastic and not hyperplastic, also by ingrowth of connective tissue. A true intra-uterine rickets is yet unproved, and thus cannot be compared, nor be completely excluded,

FŒTAL CRETINISM (Virchow).—Thyroid gland administration has no effect. Mental condition widely different.

DISTURBANCE OF AMNIOTIC PRESSURE (Jansen).—Hydramnios not infrequent.

ERROR OF AN INTERNAL SECRETION, e.g., pituitary.—No pathological evidence.

CONGENITAL SYPHILIS.—No other signs present.

Heredity and consanguinity unproved.

Bones Mainly Affected (in order of severity).—(1) Femur and humerus; (2) Tibia and ulna; (3) Base of skull. Symmetrical distribution. Radius and fibula less affected, hence articulation of joints set at abnormal angles. Bones are abnormally hard, no softening.

VII. OSTEOMALACIA.

(*Mollities Ossium.*)

Decalcification and absorption of bone occurring in adult life; the softening resulting in bending, deformities, and tendency to fracture.

Osteomalacia, *continued.*

Etiology.—

SEX.—Females predominate (92 per cent). Especially associated with *pregnancy.*

GEOGRAPHICAL DISTRIBUTION.—Foci on the Rhine and in Switzerland.

No hereditary factor. Never congenital.

Morbid Anatomy.—*Decalcification is essential change.* Also degeneration and softening of matrix and absorption.

BONES.—Very soft and light, may float.

HISTOLOGICAL CHANGES.—

1. *Compact Bone.*—(i) Haversian canals dilated. (ii) Adjacent substance free of lime salts. (iii) In still calcified tissue, bone corpuscles are large, irregular in shape, and crowded together, suggesting absorption of bone. Structure of lamellæ obscured.
2. *Medullary Cavity.*—Trabeculæ thin; little calcified bone; much osteoid tissue, some apparently newly formed by the numerous osteoblasts present.

Pone-marrow very vascular: hæmorrhages and cysts common.

No changes in ovaries.

Pathogenesis.—Phenomena connected with pregnancy and female sexual functions are undoubtedly main and usual factors; but presence of others shown by occasional occurrence in males. Note: (1) Softening of bones, especially pelvis, normally in pregnancy; (2) Osteomalacia commonly associated with, and advances in, pregnancy; (3) Parturition, abortion, or ovariotomy often arrest progress.

THEORIES.—Include: (i) Action of internal secretions, ovarian or parathyroid (latter is specially connected with calcium metabolism); (ii) Acidosis. Formerly erroneously attributed to lactic acid removing calcium. Endemic areas suggest food and water as factors.

Bones Affected.—(1) Pelvis; (2) Spine. Less markedly, thorax and extremities.

Symptoms.—

ONSET with rheumatic pains, general weakness. Then bending of legs, and waddling gait. On examination, deformities present.

WHEN DEVELOPED.—Often much deformity. Main changes:—

1. PELVIS.—Sacrum pushed forward by weight of body, and acetabula inwards by the femurs; symphysis pubis protudes like a beak. General 'clover-leaf' shape, and great narrowing of the pelvis.
2. SPINE.—Curvature often extreme.

In severe forms, bending or fractures of legs, sternum, ribs.

URINE.—Increase of calcium phosphate reported, and occurrence of renal calculi.

Progress.—Variable. May progress only in pregnancy, or be arrested by ovariotomy. Other cases advance, with death in one to ten years, usually from pulmonary diseases.

Treatment.—

IN PREGNANCY.—Decision of abortion or Cæsarean section depends on history and progress of disease and deformity of pelvis.

IF SUCKLING.—Wean child.

DRUGS.—Phosphorus pill, gr. $\frac{1}{100}$, t.d.s. increasing to gr. $\frac{1}{20}$. Lime useless.

OVARIOTOMY.—In progressing cases. Arrest not invariable.

APPENDIX.

I. DIABETES.

INSULIN.*

Insulin is the name of a specially prepared extract of pancreas, believed on adequate evidence to contain internal secretion of the islands of Langerhans. The brilliant researches on this subject have been carried out by F. G. Banting and C. H. Best, of Toronto, and their co-workers. Previous investigations had established: (1) Removal of pancreas produced diabetes; (2) No pancreatic extracts so far had any effect on diabetes. Conclusion was that an internal secretion existed, but was destroyed in extracts by proteolytic enzymes. The aim of these workers was to produce pancreatic extract without such enzymes.

Preparation of Insulin.—The main steps in researches leading to present methods have been as follows:—

1. It was known that if pancreatic ducts are ligatured, cells secreting digestive enzymes degenerate in a few weeks, while islands of Langerhans are little affected. Banting and Best prepared extracts of such glands, and found that injections into depancreatized dogs diminished sugar in blood and urine, and lengthened life. This is regarded as evidence of presence of internal secretion.
2. Fœtal pancreas contains little or no proteolytic enzyme. Extracts of such glands have had similar effects to those described in (1).
3. Alcoholic extracts of adult ox pancreas were finally employed, and present preparation is so obtained by an elaborate method.

Standardization.—Insulin lowers blood-sugar in normal rabbits. This is employed for standardization. Three *units* is the amount of insulin which on subcutaneous injection lowers percentage of blood-sugar to 0·045 within 4 hours in a rabbit weighing 2 kilo. from which food has been withheld for 16 to 24 hours. (At 0·045 per cent rabbits develop convulsions with intervals of coma, but can be revived with dextrose.) Present preparation contains 20 units in 1 c.c.

Influence on Carbohydrate Metabolism.—Action of injection studied on rabbits and depancreatized dogs. Results on human beings with diabetes are in agreement. Principal effects are:—

1. Blood-sugar falls and amount of urinary sugar diminishes.
2. Ketonuria disappears.

* *See* Macleod, *Brit. Med. Jour.* 1922, Nov. 4.

3. Respiratory quotient rises. This is evidence of metabolism of carbohydrates.
4. In depancreatized dogs glycogen in liver increases, and fat in liver and in blood diminishes. Glycogen in heart muscle falls.

Insulin injections prevent hyperglycæmia from any cause, including ether anæsthesia, except excessive doses of epinephrin.

Mode of Action.—Presence of insulin greatly increases rate of disappearance of sugar from a solution perfused through an isolated mammalian heart. This suggests that action is on muscular tissue, but investigations are not yet complete.

Insulin in Treatment of Diabetes.—Subcutaneous injections have been shown to remove manifestations of diabetes in human beings. Injections need daily repetition. Overdose may produce convulsions, but these can be controlled by injections of dextrose.

II. GASTRIC ANALYSIS.

FRACTIONAL TEST MEALS.

The method of 'fractional test meals' was introduced by Rehfuss and Hawk in 1914, and is now being extensively studied.

Technique.—

a. TUBE.—A small-bore rubber tube, about Jaques No. 6, weighted at the gastric end, and with several small holes above this, is used. Is passed on the fasting stomach, a 10-c.c. syringe is attached, and the resting juice, if any, removed.

b. TEST MEAL.—Two tablespoonfuls of breakfast oatmeal mixed with one quart of water; boiled slowly to one pint; strained through muslin. Given warm.

Give meal with tube in position. Withdraw 7 to 10 c.c. of contents through syringe every ¼-hour until stomach is empty. The fractions are analysed by any of the accepted methods, e.g., Töpfer's reagent and phenolphthalein, or Volhardt's. Results obtained are plotted as a curve.

Examine also for: (1) Bile—evidence of duodenal regurgitation; (2) Starch—evidence of digestion; (3) Blood and mucus.

Results.—Information is afforded concerning: (1) Secretory functions—by measure of acidity; (2) Motor functions—by time of emptying.

NORMAL DIGESTION.—Amount of fasting juice slight and acidity low. Acidity rises to maximum in 1 to 1½ hours (figures as in Ewald's meal); then remains fairly constant, or falls slightly. Stomach empty in 2½ to 3 hours. Starch disappears in 1½ to 2 hours. Bile may appear in third hour.

DUODENAL AND PYLORIC ULCER.—Resting juice may be of high acidity, but is not invariably so. After initial fall on introduction of meal, acidity rises sharply: maximum in 1 to 1½ hours.

A steep fall may occur with presence of bile, due to alkaline duodenal contents. Stomach often empties very rapidly.

PYLORIC STENOSIS.—Resting juice often of low acidity. Rise of acidity may be slow, but to high level, maximum sometimes in third hour.

Note.—Presence of bile in contents within 3 hours is evidence against stenosis.

III. JAUNDICE.

VAN DEN BERGH'S TEST.*

By the use of Van den Bergh's test differentiation of the two main types of jaundice can be effected, viz., toxic (including catarrhal) and obstructive.

Van den Bergh discovered :—

1. That Ehrlich's diazo reagent is a very sensitive test for bilirubin in blood serum.
2. That the reaction differs in the two types of jaundice.

No substance other than bilirubin gives a positive test.

Technique.—

REAGENT.—Two solutions which keep well :—

A.	Sulphanilic acid	1 c.c.
	Concentrated HCl..	15 c.c.
	Distilled water	1000 c.c.
B.	Sodium nitrite	0·5 grm.
	Distilled water	100 c.c.

The solutions are mixed in the proportion 25 c.c. of solution *A* to 0·75 c.c. of solution *B* immediately previous to use.

BLOOD SERUM.—Remove 10 c.c. of blood from vein, allow to clot, and pipette off separated serum.

Performance of Test.—

1. IMMEDIATE OR DIRECT REACTION.—Add 1 c.c. of reagent to 1 c.c. of serum in a small test-tube. If test is positive, a bluish-violet colour develops and is at maximum in 10 to 30 seconds. Intensity depends on the amount of bilirubn.
2. INDIRECT REACTION.—If direct reaction is negative, the indirect test is performed as follows : To 1 c.c. serum add 2 c.c. of 96 per cent alcohol ; centrifuge ; pipette off 1 c.c. of clear supernatant fluid, and add to it 0·5 c.c. of alcohol and 0·25 c.c. of diazo reagent. If test is positive, a violet-red colour develops and is maximum instantly.

Other changes which may occur are :—

DELAYED REACTION IN DIRECT TEST.—A reddish colour develops in 1 to 15 minutes ; is of same significance as indirect reaction.

BI-PHASIC REACTION.—Reddish colour appearing immediately, and gradually deepening to violet.

* *See* McNee, *Brit. Med. Jour.* 1922, May 6.

Interpretation of Results.—

DIRECT REACTION POSITIVE.—This indicates obstructive jaundice.

INDIRECT REACTION POSITIVE WITH NEGATIVE DIRECT REACTION.—This indicates toxic jaundice (including catarrhal).

Notes.—(1) If direct reaction be positive, indirect is necessarily also positive. (2) The meaning of the bi-phasic reaction is at present uncertain. (3) A quantitive measure of the reaction can be carried out by comparison with a standard solution of iron sulphocyanide.

The difference in the two tests probably depends on bilirubin in toxic jaundice being loosely combined with protein in the serum, liberation occurring under the action of alcohol or with time.

IV. TRYPANOSOMIASIS.

BAYER 205.

Bayer 205 is an organic compound containing amino-naphthalene-sulphonic acid. Animal experiments prove that it is a powerful trypanocide, and also that a single dose confers immunity to infection for considerable periods. It is being extensively tried in human trypanosomiasis.

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